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PHYSIOLOGICAL EFFECTS OF "SOCIAL STRESS" IN WILD RATS—I

THE ADRENAL CORTEX*

S. A. BARNETT

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1. INTRODUCTION

AN experimental study has recently been made of the social behaviour of wild rats, *Rattus norvegicus* Berkenhout (BARNETT, 1958a)†. This, the common 'brown' rat of Europe, is the species to which all tame laboratory rats belong, but the behaviour of tame rats differs in many ways, apart from the obvious ones, from that of wild ones. Special attention was paid to the circumstances which evoke fighting in this species. It was shown that groups of adult male rats, initially strangers to each other, may live together in large cages with little or no strife, provided that none has had experience of the cage before the beginning of the experiment. By contrast, if females are present, most of the males lose weight and many die. If an adult male is put in an established colony, whether all-male or mixed, at least one of the resident males attacks it; usually the newcomer dies.

It has also been briefly reported that the adrenal glands of males in the mixed colonies are larger than those of males in all-male colonies or of controls kept in small cages; and that the adrenal cortex of a male which has been acutely 'stressed' by introduction into a colony of strangers may have a reduced content of sudanophil substance, presumably indicating an exceptionally high output of cortical hormones (BARNETT, 1955, 1956).

The present paper describes the decline and death of male rats subject to attack by other males, and gives in detail the observations made on adrenal glands from rats used in the experiments on behaviour.

2. RATS AND METHODS

2.1. *The rats*

The rats were bred in the laboratory from wild *R. norvegicus* trapped in Glasgow. These rats, kept in small cages alone or in male–female pairs, were healthy, grew well and, in many instances, bred well.

The rats, both experimental and control, had to be handled when they were weaned at 4 weeks, and when weighed and transferred to the experimental cages. (Further information on their management is given by BARNETT, 1958a.) This handling had no detectable ill-effect on any of the rats used in these experiments. A rat seized by the experimenter squealed, struggled and bit; sometimes it escaped, and fled when pursued; but, once released into a cage, it showed no signs of 'shock'. KING (1939) mentions shock from handling in some of her wild rats, but gives no details. RICHTER (1957) also reports death in wild rats evidently due to handling. Despite this, it did not occur in the rats used in these experiments, possibly because of familiarity with the presence of human beings.

* From the Department of Zoology, Glasgow University, Scotland.

† In the account which follows, this paper is referred to by its date only, (1958a).

2.2. *Methods*

The data presented in this paper are from two types of experiment; the behaviour of rats in both types has been fully described elsewhere (1958a).

(i) Adult rats, strange to each other, were weighed and earmarked, and placed together in a large, unfamiliar cage with food ('diet 41'), water and nest boxes; some colonies were exclusively male, others included females. After 7–22 weeks the surviving males were removed, killed by a blow on the head and weighed; they were inspected for wounding and for gross indications of disease, and their adrenal and thyroid glands were removed. (ii) Adult male rats were also added, usually one at a time, to established mixed or all-♂ colonies; in six instances the 'colony' consisted of only one resident male. Most of these interlopers were left until they were dead or at the point of death (or, in two instances, until they were absorbed into the colony); but 3 interlopers were removed, when still in a healthy state, after 48 hr exposure to attack, and killed by the experimenter.

The left adrenal and the thyroid gland of some of the rats from each type of experiment were fixed in Bouin's fluid, the right adrenal in formalin calcium. Adherent fat was removed from the adrenals before fixation; after 24 hr fixation the adrenals were taken from the fixatives, rapidly dried on filter paper, further cleaned if necessary, and weighed on a torsion balance to the nearest 0·1 mg. They were then returned to the fixatives, and the right adrenals were later embedded in gelatin, sectioned and stained with sudan black. Several such sections were examined from each gland. Left adrenals were embedded in paraffin, sectioned and stained with haematoxylin and eosin. Observations on the thyroids will be reported separately.

The data on adrenal weights, given in Tables 2 and 3, were all taken from rats which had been born in the laboratory. The males of each litter used were divided between ♂–♀ colonies, all-♂ colonies and small cages each containing 1 male and 1 female. The colonies used for this purpose are listed in Table 1. Data on adrenal weights of males from ♂–♀ colonies were taken only from

TABLE 1.—COLONIES WHICH PROVIDED DATA ON ADRENAL WEIGHTS

	Colony No.	Number of males	Males died	Duration (weeks)
Male-female colonies	3	6	3	14
	5	10	4	7
	6	5	1	9
	7	6	2	13
All-male	1	6	0	11
	9	5	0	22
	33	7	0	8

TABLE 2.—RELATION OF ADRENAL WEIGHT TO BODY WEIGHT

Body-wt. (g)	Number of rats	Mean adrenal wt. (mg)	Standard deviation
201–40	3	62·3	7·01
241–80	7	55·6	12·63
281–320	8	61·5	16·06
321–60	8	63·8	16·27
361–400	3	66·2	19·82
401–40	1	72·5	–
All rats	30	61·8	14·73

those that survived the whole period during which the colony was maintained. A few of the males from the colonies were not killed at the end of the experiment, and so their adrenal weights are not available.

TABLE 3.—ADRENAL WEIGHTS

	Number of rats	Range of body weights (g)	Mean wt. of adrenals (mg)	Mean wt. of adrenals (mg/100 g)
Controls*	15	205–375	60·0 ± 4·09	20·7 ± 1·46
All-♂ colonies	15	210–405	63·6 ± 3·42	20·6 ± 1·50
♂–♀ colonies	15	180–430	82·5 ± 5·32	30·3 ± 2·43

* The controls were rats kept in small cages, each alone with one female.

3. DEATH

The first effect of attack by a fiercer or more powerful male was either flight or the adoption of a defensive posture. After a vigorous bout of fighting the inferior male often lay prostrate, panting irregularly (Fig. 1); but apparently complete recovery from this state occurred in 10–30 min, provided there was no further attack during that period. Similar recovery, from another type of stress, has been described by RICHTER (1957).

Rats which had been subjected to severe and persistent attack by a dominant male had (with some exceptions) a characteristic appearance: their fur was staring or bedraggled; movements were slow and feeble; typical, thorough grooming was not carried out; and their posture gave them an appearance of 'dejection'. Such 'omega' rats tended to flinch at the approach of any other rat, even a female. Most subordinate rats lost weight (1958a; see also Table 4). Rats did not eat on first entering a strange cage, whether residents were present or not (1958a), but the general decline in weight of 'omegas' cannot be attributed to anorexia: those which survived persecution for more than 1 or 2 days ate readily, and often rapidly, when they had the opportunity. They were frequently disturbed while eating by the approach of a dominant male, but even this is not the whole story: in all colonies in which there was conflict between males, food cubes were accumulated in the nest boxes, and so a good deal of feeding could go on without interference.

The account in the previous paragraph does not apply to certain rats which adapted themselves to a subordinate role. These, called 'betas' (1958a), not only survived, but increased in weight.

Many of the rats that died were unwounded, and the damage to those that were injured was only superficial: it included gashes on the limbs, ear and tail, and less often in the skin of other parts of the body. This applied even to those whose death occurred many days after exposure to attack.

Among colony members, death occurred at any time from the second day of the experiment to the tenth week, but the great majority of deaths occurred within 3 weeks of the beginning of the experiment. Of 27 males put in already established colonies ('interlopers'), 6 died within 24 hr, 11 on the second to seventh days, 5 after more than seven days and 2 survived. The remaining 3 were killed by the experimenter after 48 hr exposure to attack by a single resident male. All those that died within 24 hr were victims of one exceptionally ferocious male.

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TABLE 4.—INDIVIDUAL BODY AND ADRENAL WEIGHTS

Rat No.	Initial wt. (g)	Final wt. (g)	Adrenals wt. (mg)			Exposed (days)
Interlopers	w_1	w_2	a	$\frac{a}{w_1} \cdot 100$	$\frac{a}{w_2} \cdot 100$	
44	350	305	107·5	30·7	35·2	12
47	400	340	162·0	40·5	47·6	1
64	350	320	111·6	31·9	34·9	1
65	325	285	119·6	36·8	42·0	8
101	290	245	80·9	27·9	33·0	2
103	305	275	89·1	29·2	32·4	2
104	320	305	106·3	33·2	34·9	2
'Omegas'						
35	250	170	74·2	29·7	43·6	10
36	245	215	103·0	42·0	47·9	11
74	350	300	89·2	25·5	29·7	4
75	290	215	122·8	42·3	57·1	9
76	380	290	111·3	29·3	38·4	10
77	380	275	84·8	22·3	30·8	13
'Alphas'						
37	280	290	72·0	25·7	25·2	20
78	355	420	107·9	30·4	25·7	13
102	—	415	64·6	—	15·6	—
105	—	240	108·2	—	45·1	—
109	—	350	52·2*	—	14·9*	—
63	325	285	119·6	36·8	42·0	8

* Pathological changes in the left adrenal make this figure misleading.

4. ADRENAL WEIGHTS

4.1. *In relation to body weight*

In prolonged 'stress' the adrenal glands of mammals tend to enlarge (SELYE, 1947). The noxious stimuli which produce this effect are very diverse: they include overcrowding, and conditions which induce 'fear' or 'anxiety' (RICHTER, 1949; CLARKE, 1953; BARNETT, 1955, 1956; CHRISTIAN, 1955; BLISS *et al.*, 1956; CHRISTIAN and DAVIS, 1956).

The form in which adrenal weight data are most usefully presented depends on the relation of adrenal weight to total body weight or to other measures of the mass of metabolizing tissue. DONALDSON (1924) gives figures for recently trapped wild rats. Calculation from his data gives a mean of 80·5 mg for total adrenal weights from 48 males above 150 g body weight; (there is no information on variability). DONALDSON'S figures are given by body-weight groups, and suggest that, above 150 g body weight, adrenal weight is independent of body weight.

This has been tested on 30 of the rats used in the present experiments (Table 2): 15 were controls, kept in small cages, and 15 were from all-♂ colonies; statistically, these formed a single homogeneous group. The mean adrenal weight was 61·8 mg. Calculation of the F ratio (SNEDECOR, 1946) showed no evidence from these data of a relationship between body weight and adrenal weight. The relationship between

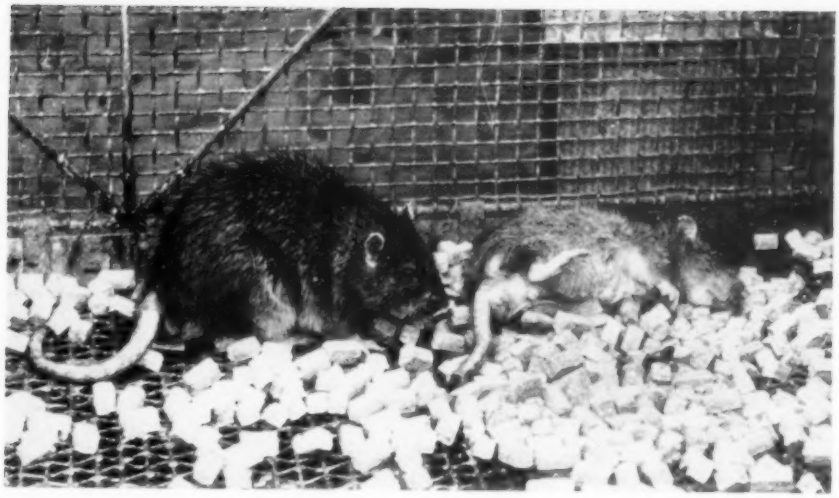

FIG. 1.—An 'interloper' collapses during attack by a resident male. The resident still shows pilo-erection, but ceases to pay attention to the interloper while the latter is immobile.

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log body weight and log adrenal weight was investigated in the same way (*cf.* CHRISTIAN, 1953), but again each was independent of the other on the evidence of these data. CHRISTIAN and DAVIS (1955) found a linear relationship between adrenal weight and body length; they do not give the actual adrenal weights of the rats they studied and, since body lengths were not recorded in the experiments described here, their results cannot be compared with the present ones.

4.2. *Of rats in undisturbed colonies*

Table 3 gives mean adrenal weights of male rats from ♂–♀ colonies, all-♂ colonies and from small cages. Most of the members of the first group lost body weight, while most of those in all-♂ colonies, and all the control rats in small cages, gained (1958a). The relative adrenal weights of the first group have therefore been related to the body weights at the beginning of the experiment, those of the controls and of rats from the all-♂ colonies to body weight at the end of the experiment. However, as has been shown in the previous section, relative adrenal weights within this range of body weights are less important than absolute weights. The figures indicate an enlargement of the adrenals in the males from ♂–♀ colonies; the difference from the control figure is statistically significant ($P \leqslant 0{\cdot}05$). This is in conformity with the observation (1958a) of greater conflict in these colonies. The mean adrenal weight of the rats from the all-male colonies was, as already indicated, not significantly higher than that of the control rats.

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The mean weight of the adrenals from the ♂–♀ colonies was very close to the figures given by DONALDSON (1924) and by WOODS (1957a) for recently trapped wild males. The data of Table 3 are from survivors only. Table 4 (discussed in detail in § 4.3 below) includes the adrenal weights of 8 further members of mixed colonies. Six of these were 'omega' or defeated subordinate animals (1958a) which died after 4–13 days in the colony; the other 2 were dominant (alpha) rats, neither of which was in conditions of conflict for more than 13 days. These form, therefore, a distinct group from that of the males from ♂–♀ colonies referred to in Table 3. The mean adrenal weight for this group of 8 rats was $96{\cdot}65 \pm 6{\cdot}08$ mg, a figure markedly higher than DONALDSON'S and significantly higher ($P \leqslant 0{\cdot}05$) than that for males from ♂–♀ colonies given in Table 3.

There was much individual variation in behaviour among the rats from which the data of Table 3 are taken. In particular, some of the males in the ♂–♀ colonies were socially dominant, others were subordinate (1958a). It might be thought that the adrenal weights of these two classes of males would differ; but, when the rats which gained in weight were compared with those that lost, no difference emerged: adrenal enlargement evidently occurred in both groups. This fact is further illustrated in Table 4.

4.3. *Of individual rats subjected to 'stress'*

Table 4 gives individual body and adrenal weights for 19 of the rats on which detailed observations of behaviour were made. All these rats were subjected to stress in the sense that they were involved in conflict with other males. 13 of them died evidently as a result of this stress.

The first 7 in the list were added to established colonies containing one or more resident males. The adrenal weights of the rats of this group were all high. No. 65

was put in an established all-♂ colony and died after 8 days; its adrenal weight was very high. Nos. 101, 103 and 104 were each put separately in a cage containing a single resident male, and were left there for only 48 hr; the first two were still vigorous at the end of this time, and were killed by the experimenter, but No. 104 died just before the period had elapsed. All three, but especially 104, had large adrenals.

Nos. 44, 47 and 64 all had complex histories. No. 44 was originally a member of a colony in which it had a role subordinate to that of a large and fierce dominant male; it survived a period of persecution by this 'alpha' male, and settled down to a 'beta' status (1958a). Later, still in excellent health, it was added to another, ♂–♀ colony, in which it survived for 12 days. Its relative adrenal weight was high.

Nos. 47 and 64 had both been dominant rats in colonies containing both males and females. Each was later put in a strange ♂–♀ colony, and each survived only for 1 day in these conditions. Both had relatively large adrenals; this applies especially to No. 47, which was a particularly fierce individual (the one responsible for the early death of 6 interlopers, as described in § 3).

The second group of rats in Table 3 consists of the 6 'omega' males from two ♂–♀ colonies already referred to in § 4.2 above. In each of these colonies there was a single dominant male which survived, while all the other males died. All these rats had adrenal weights above the mean of the controls, and all, except No.35, above the mean of the rats from ♂–♀ colonies given in Table 3.

The third group consists of rats which were never defeated in battle: they were involved in much fighting, but only with 'inferiors.' No. 37 was the dominant male of a group of 3 males and 3 females; (the other 2 males died as a result of its attacks); at the end of 20 days, 9 days after the death of the second of the 2 defeated males, it was killed, while still healthy, by the experimenter; its adrenal weight showed only about 20 per cent excess over the mean of the controls. Nos. 102, 105 and 109 were born in the laboratory, and were each separately mated with a single female with which they shared a small cage. At the age of about 1 year each was put alone in a large cage, into which strange males were singly introduced from time to time. Each of these strange males was attacked and defeated. All 3 of these rats, while still evidently healthy, were killed by the experimenter. The adrenal weight of No. 102 was near the control mean; that of No. 105 was high The left adrenal of No. 109 had undergone pathological changes, and weighed only 15·9 mg; the right adrenal weighed 36·3 mg. This is obviously a heterogeneous group, and no generalization can be made about it.

Finally, No. 63 was a rat from a mixed colony which displayed a 'compulsion' to attack other males, even if they were larger and stronger; this rat wore itself out in two days. Its adrenals were large.

5. ADRENAL HISTOLOGY

5.1. *Evidence of increased activity*

Increased secretory activity of the adrenal cortex may be inferred from any one of three aspects of its appearance (reviewed by CHESTER JONES, 1957).

(i) After treatment with a sudan stain the size of the lipid droplets in the cells of the zona fasciculata may be studied: fine droplets indicate high activity, by contrast with the large droplets which fill the cells of the 'normal' gland. (ii) Intense stimulation can lead to the reduction or almost complete disappearance of the sudanophil material from these cells. (iii) Increased activity is accompanied by enlargement of the cells of the zona fasciculata: this hypertrophy is responsible,

largely if not entirely, for the increased weight of the adrenals in stress (POPJÁK, 1944; ROGERS and RICHTER, 1948); POPJÁK showed also that an increase of 31–42 per cent in relative adrenal weight can occur in stressed albino rats in only 24 hr. In sudan preparations hypertrophy tends to produce a 'bubbly' and disorganized appearance (Figs. 8, 9, 10), instead of the orderly columns of cells typical of the zona fasciculata (*e.g.* Figs. 2, 3, 4); in H & E preparations the cells are visibly swollen, and the way in which the columns have been disrupted can be readily seen. Other changes than these take place in the hyperactive adrenal cortex, especially in the other two zones, but these were not found useful indices of activity in the present study; the reaction of the glomerular zone was particularly unpredictable.

Table 5 summarizes the histological observations made on 54 rats. The adrenals are classified by the appearance of the zona fasciculata in sudan preparations. Those called 'normal' were heavily and evenly impregnated with the stain throughout the zona fasciculata and the lipid droplets were large (Figs. 2, 8); consequently, this group included hypertrophied adrenals. 'Active' glands had finer lipid droplets, and some

TABLE 5.—STATE OF ADRENAL CORTICAL LIPID

	Number of rats	Normal	Active	Grossly depleted
Controls	15	15	0	0
All-♂ colonies	15	7	8	0
♂–♀ colonies	12	2	10	0
Interlopers	12	2	2	8

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of the cells had an obviously reduced amount of sudanophil material (Figs. 3, 4). Finally, in some adrenals there was an almost uniform and great reduction in the amount of sudanophil material; these are called 'grossly depleted' (Figs. 5, 6, 7).

All the control rats had normal glands. CAIN and HARRISON (1950) found evidence of a cycle of depletion and recovery in the adrenals of laboratory albino rats, but there is no reason to think that this occurs in wild rats: it has long been known that the adrenal glands of wild rats are much larger, both absolutely and relative to body weight, than those of tame ones (see ROGERS and RICHTER, 1948), and it may be supposed that they are less readily depleted (see also MOSIER, 1957).

Of the 15 rats from all-♂ colonies, 7 had normal and 8 had active adrenals. There was no evidence of a relationship between activity, inferred from histological appearance, and adrenal weight. Since there was no indication of enlargement from the weights of the adrenal glands of this group, it might seem surprising that signs of activity were found: in tame laboratory rats increased secretory activity of the adrenal cortex is accompanied by hypertrophy (*cf.* CHESTER JONES, 1957). Perhaps, again, the greater size of the wild adrenal allows some degree of hyperactivity without hypertrophy; if so, the state of the cortical lipid in wild rats may be regarded as a more sensitive index of cortical function than adrenal weight.

In the ♂–♀ colonies, 10 of the 12 rats whose adrenals were examined showed cortical hyperactivity. The 2 adrenals which showed no sign of depletion or of reduction in lipid droplet size were markedly hypertrophic: both sudan-stained sections and those treated with H & E displayed swollen cells in the zona fasciculata.

(Figs. 8, 9) The weights of the adrenals of rats from ♂-♀ colonies, as described in § 4.2 above, indicated a general tendency to enlargement; it seems possible that, in the two exceptional rats of this group, hypertrophy had gone so far that it had permitted the re-accumulation of lipid despite the continued stress.

The last group consisted of 12 interlopers—rats which, on being put in a strange cage, had been attacked by resident males. Of these, 10 had adrenals which showed evidence of additional activity; and in 8 the zona fasciculata was grossly depleted (Figs. 5, 6, 7). The two which fell into the 'active' category both had unusual histories. One had evidently adapted itself to conditions in the colony: it survived for 46 days after it was put in the cage, and was healthy when it was removed and killed by the experimenter; it had grown during the experiment. The other (No. 47) had been a dominant male in another colony; its adrenals were the heaviest recorded (Table 4), and histologically showed conspicuous hypertrophy, yet at the same time the lipid had the appearance characteristic of an active gland.

The 2 interlopers of which the glands were histologically normal died within a few hours of being attacked by an exceptionally fierce male.

6. DISCUSSION

6.1. *The adrenal changes*

CHRISTIAN (1955, 1956) and CHRISTIAN and DAVIS (1955, 1956) found that adrenal weight increases with crowding in populations of tame and wild-type mice (*Mus musculus*) and of wild *Rattus norvegicus*. Comparable observations have been made on voles: CLARKE (1953) described enlargement of the adrenal glands resulting from fighting in *Microtus agrestis*, and LOUCH (1956) found evidence of increased adrenal cortical activity in dense populations of *M. pennsylvanicus*.

The facts given in §§ 4.2 and 4.3 above suggest that in wild male *R. norvegicus* the enlargement is due to continual conflict involving all the males of a colony. The effect on adrenal weight is observed both in dominant (alpha) and in subordinate (beta and omega) rats: the largest adrenals recorded belonged to the fiercest male (No. 47, Table 4), but the second largest to an interloper (No. 75) exposed to attack for 9 days. In the account, already published (1958a), of the behaviour of these rats, detailed evidence is given for regarding the fighting of males as essentially territorial: that is, it consists of attack on all (strange) males encountered within a defined area (in the experiments, the cage). This is obvious where the fighting is against an interloper, but evidence is also given that even the extra fighting resulting from the presence of females is primarily territorial; it is not fighting for the females.

The difference between the ♂-♀ and the all-♂ colonies shows that crowding in itself is not the cause of conflict; this is more fully demonstrated in the previous paper (1958a): in particular, colonies of litter mates of both sexes are described, in which there was no conflict.

CHRISTIAN, and CHRISTIAN and DAVIS, in the papers quoted above, found adrenal enlargement also in the females of dense populations. In general, females do not fight, except in defence of a nest containing young (1958a). It seems likely that in dense populations greater demands are put on females in this respect; there are also no doubt more frequent attempts at coitus by males. These two factors may account for adrenal hypertrophy in females, but there is no direct evidence on this point.

In males persistent, vigorous and energetic attack usually produced extensive loss

FIGS. 2–9 are photomicrographs of sections of the right adrenals of male rats. The stain is sudan black.

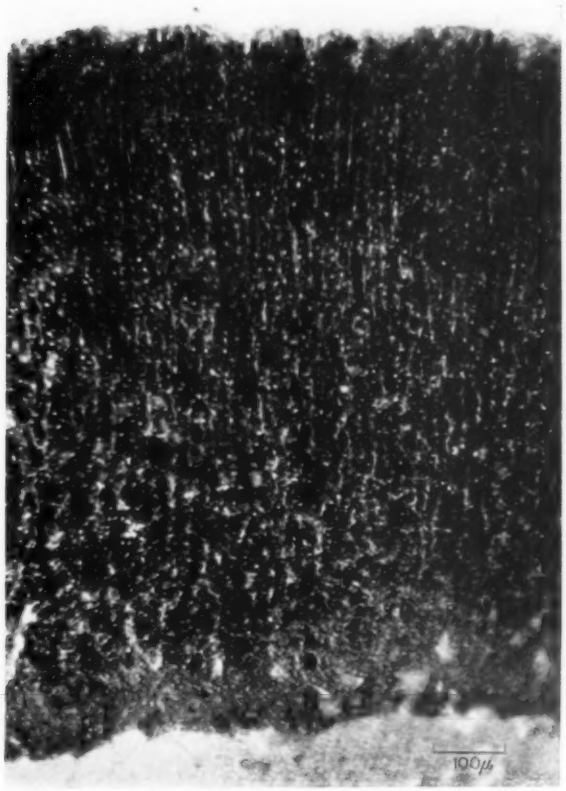

FIG. 2.—Section of adrenal of control rat. The whole of the cortex is stained; only the medulla (bottom of picture) is not darkened.

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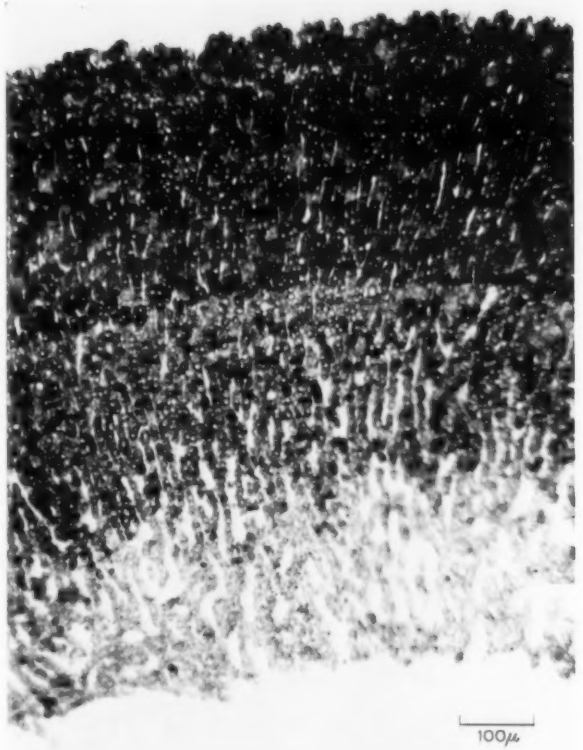

FIG. 3.—Section of 'active' adrenal. The rat had been persistently persecuted, but was in good health when killed. There is uniform, moderate depletion in the inner part of the zona fasciculata; in the outer part there are small patches of depletion. The zona reticularis is only lightly stained, and the medulla (bottom of picture) not at all.

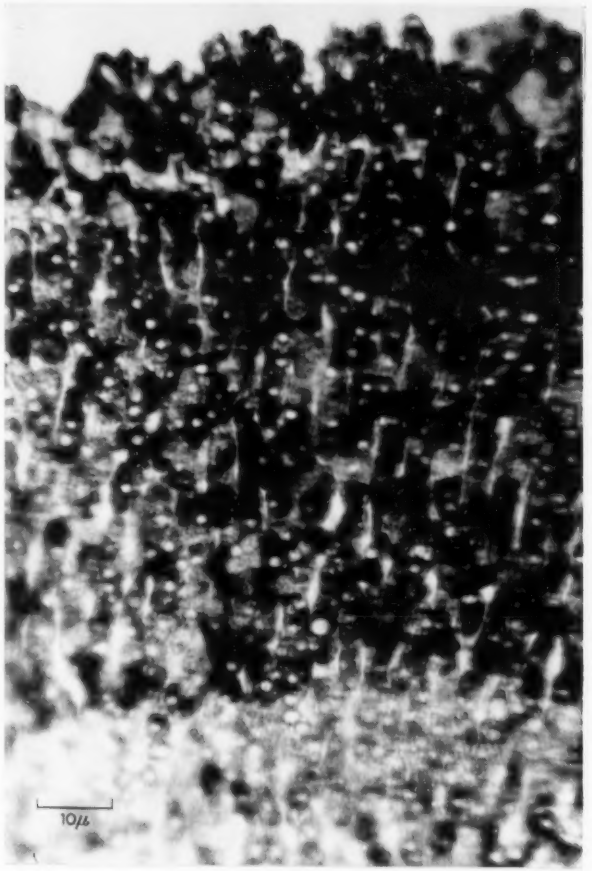

FIG. 4.—As 3. The patchy depletion of the lipid in the outer fasciculata is shown, and the clearly marked boundary of the inner fasciculata. The zona glomerulosa (at the top) is full of lipid, but the narrow transitional zone below it is almost unstained.

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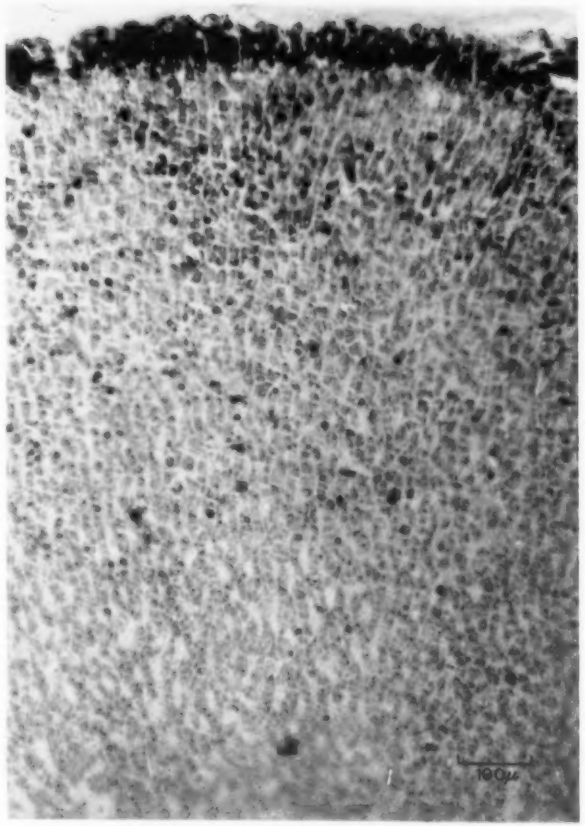

FIG. 5.—Section of grossly depleted adrenal. This rat was an 'interloper' which died within 14 hr of introduction into the experimental cage. The adrenals were removed very shortly after death. The zona glomerulosa (at the top) has retained its lipid, but little remains in the fasciculata.

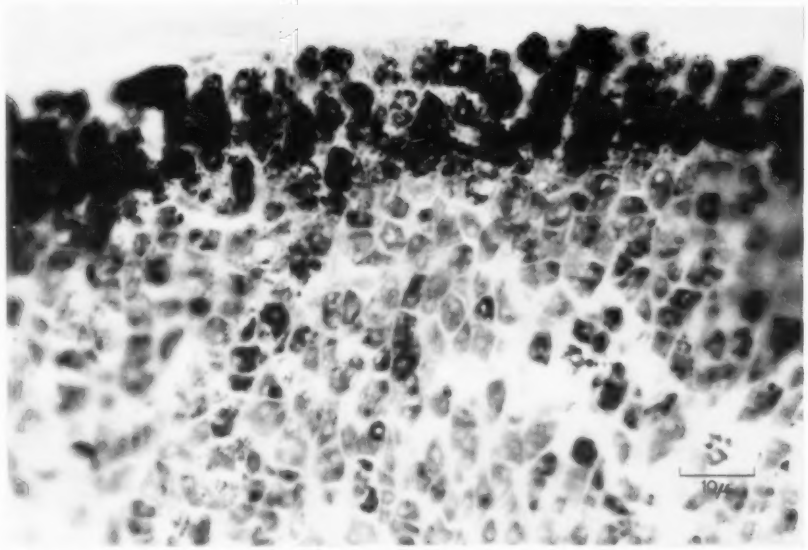

FIG. 6.—As 5. The fine granules of what lipid remains in the fasciculata can be seen; there is also some evidence of depletion in the glomerulosa.

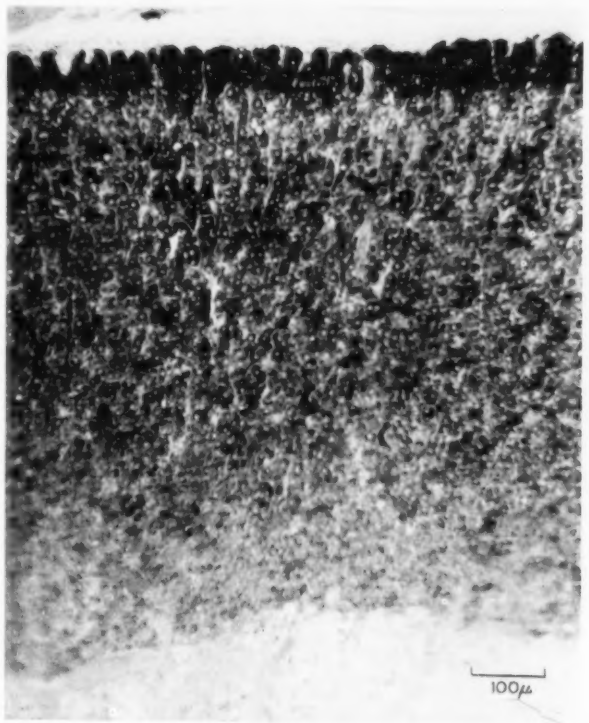

FIG. 7.—Section of grossly depleted adrenal. The rat (No. 44, Table 4) had been a 'beta' (a successful subordinate male) in one colony. Later it was introduced as an interloper into another colony; 12 days later it was removed and killed, when at the point of death. The glomerulosa (at the top) has retained its lipid, but most of that in the fasciculata has been lost and the cells of this zone are also enlarged.

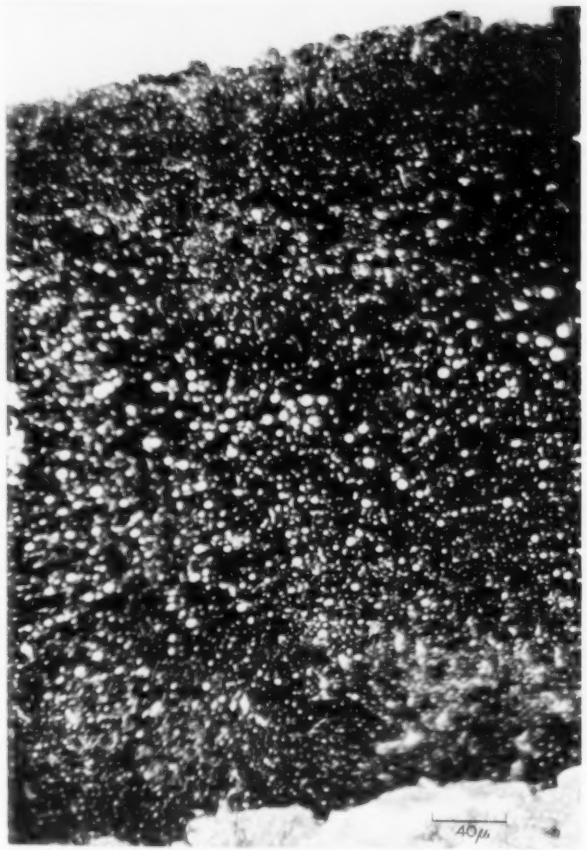

FIG. 8.—Section of grossly hypertrophied adrenal. The rat had been in a ♂–♀ colony for 13 weeks, during which it had lost 25 per cent of its body weight. The adrenal weight (106·6 mg) was high. There is no lipid depletion, but great enlargement of the cells of the zona fasciculata, with consequent disruption of the cell columns. The medulla (unstained) is at the bottom.

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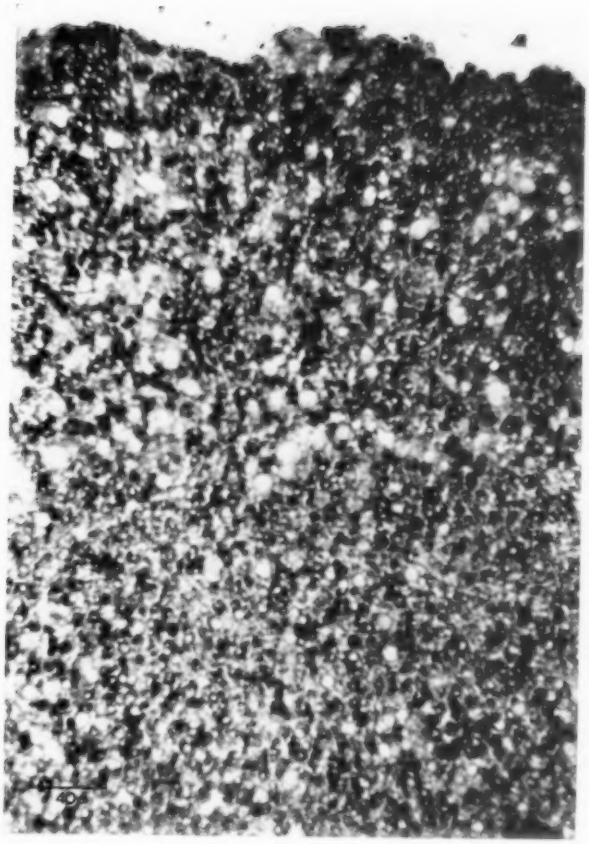

FIG. 9.—Section of grossly hypertrophied adrenal. This was from an exceptionally fierce rat (No. 47, Table 4) which had been an 'alpha' in one colony. Later it was added as an interloper to another ♂–♀ colony. After 24 hr it was clearly dying, and was removed and killed. Despite the obvious enlargement of the cells of the fasciculata, there was also lipid depletion. The glomerulosa (at the top) also shows some depletion.

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of lipid from the zona fasciculata of the attacked rat; this was observed only in 'interlopers' faced with one or more resident males in a strange cage. WOODS (1957a,b) found no cortical depletion in wild rats subjected to various kinds of stress, including 'fighting' for 30 min in a cage in which mild electric shocks were administered at 5 sec intervals. The absence of depletion in his experiments emphasizes the intensity of the stimulation to which the hypothalamic–pituitary–adrenal system of interloper males must have been subjected. The same conclusion is suggested by the fact, reported above (§ 4.3) that 2 formerly dominant rats with very hypertrophic adrenals each died, after 24 hr in a strange colony, with their adrenals showing signs of depletion.

The less severe conditions of ♂–♀ colonies led to increased adrenal cortical activity, again shown by the state of the lipid; this was accompanied, where the continued conflict was severe enough, by adrenal enlargement. (There may have been also a temporary depletion in some rats in the ♂–♀ colonies, but this was not observed.) The enlargement was partly or wholly a consequence of hypertrophy (increase in cell size) in the zona fasciculata. Thus the changes in the adrenal glands constitute an index of the degree of stress to which each rat was subjected.

These facts do not make it clear just what relation holds between adrenal cortical activity and behaviour. YEAKEL and RHOADES (1941) found genetical variation in adrenal size between different strains of tame rats, and reported that those with larger adrenals were more 'emotional.' It can, however, hardly be supposed that this difference in behaviour is caused, in any simple way, by the larger glands, or that the still larger adrenals of wild rats are responsible for their wildness and their propensity for fighting. Clearly, regardless of the particular genotype, 'stress' of various kinds leads to enlarged adrenals; this is evidently part of a physiological adaptation making possible greater resistance to stress. The genetically larger adrenals of wild rats may be assumed to fit them better for the stresses of life in natural conditions, than would the small ones of tame rats. WOODS (1957b), on the basis of the work quoted above, raises the question whether in wild, as distinct from domestic, mammals, 'metabolic depletion' of the adrenal cortex ever occurs. This question had already been answered for the wild rat (BARNETT, 1955); and the results reported above show that, while depletion is difficult to produce, it can occur as a result of physiological stimulation of the adrenal, and not only (as in WOOD's experiments) through injection of ACTH. It is possible that the rats used in the present experiments were more susceptible to depletion than those of WOODS, because they had been in captivity from birth; but this question cannot be settled on present evidence.

The great variability in adrenal weight among wild rats no doubt reflects in part the genetical variability which is to be expected in a natural population, but it may in part be also a result of differences in the conditions of life of different individuals. It is further possible that adrenal size is indirectly influenced by variations in 'personality' (1958a), regardless of the extent to which these are genetically or environmentally determined: aggressiveness or excitability, springing from a particular neural organization, may lead to behaviour which almost inevitably brings the individual into stressful situations.

The genotype-environment interaction controlling adrenal function is obviously exceedingly complex and, for genetically diverse wild rats especially, is likely to be unravelled only by further, elaborate experiment.

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6.2. *The causes of death*

The most general question raised by these experiments is the exact nature of the causes of death. This is discussed more fully elsewhere (BARNETT, 1958b). GREEN, LARSON and BELL (1939) have described death in the snowshoe hare, *Lepus americanus*, due to sudden stress; FRANK (1953) has studied mortality in crowded populations of the vole, *Microtus arvalis*. In both, there was hypoglycaemia. It is possible that death in attacked wild rats is a result of disturbed carbohydrate metabolism, related to a failure in the secretion of corticosteroids.

A quite different possibility, perhaps especially applicable to cases of death after only short exposure to attack, is that of vagal syncope resulting from excessive parasympathetic stimulation. This has been reported by RICHTER (1957) in wild rats subjected to a quite different sort of stress.

These hypotheses are readily testable by experiment. A more difficult problem is why attack by a congener should have such a specific and drastic physiological effect. At present it is possible only to speculate on this question, and that has been done elsewhere (BARNETT, 1958b).

SUMMARY

Wild rats (*Rattus norvegicus* Berkenhout) were kept (i) in all-male groups in large cages, (ii) in groups including both males and females in large cages, (iii) in male–female pairs in small cages (controls). In some experiments males ('interlopers') were added, one at a time, to established colonies. Control males, and those in all-male groups, survived well; there was a high death rate in male–female colonies; and most interlopers died.

Some rats, subjected to severe attack, died in a few hours, others only after many days. Death was not due to wounding.

Adrenal weights of control males and of males from all-male colonies were not significantly different. Those of males from male–female colonies were significantly higher. The adrenal weights of interlopers were also high. The increase in adrenal weight was not confined to rats defeated in battle: dominant rats also had large adrenals.

Staining the adrenal cortical lipid with sudan black indicated that some at least of the rats from all-male colonies had hyperactive adrenals. All the males from male–female colonies showed either signs of hyperactivity in the zona fasciculata, or gross hypertrophy. Of 12 interlopers, 8 had the adrenal cortex grossly depleted of lipid.

Some rats, subjected to severe attack, died in a few hours, others only after many days. Death was not due to wounding: it may have resulted from disturbed carbohydrate metabolism or from 'vagal syncope'. There is no explanation of the fact that attack by a congener produces such a drastic physiological disturbance.

Acknowledgement—I am very grateful to MARGARET DOYLE for her help with the histological preparations.

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THE INFLUENCE OF AGE AND FUNCTIONAL NEUROPSYCHIATRIC DISORDERS ON SYMPATHETIC AND PARASYMPATHETIC FUNCTIONS*

R. NELSON and E. GELLHORN†

(*Received* 20 *August*, 1957)

THAT changes, due to ageing, occur in the central nervous system in general and in the autonomic system in particular has been known since CHARCOT's lectures (1881). From physiologic measurements of blood sugar, temperature and acid base balance, as well as adaptation of the circulatory and respiratory system to emergencies, CANNON (1942) stated that the preservation of stability is impaired and homeostasis is maintained within a narrower range in the aged. Agreement with this statement is found in other studies. NORRIS *et al.* (1953) demonstrated a slower compensatory response in the systolic and diastolic blood pressure of older people when subjected to tilting from the horizontal and vertical planes. Older subjects have also shown a distinct decline in pulse rate reaction following atropine injection (CRAWFORD, 1924), along with a significant depression in tolerance to intravenous glucose (SCHNEIBERG and FINESTONE, 1952) when compared with younger subjects.

Experimental studies in animals have presented evidence of decline in function of the autonomic nervous system. In older rats, SAFFORD and GELLHORN (1945) found a diminished excitability of the sympathetico-adrenal system when measured by the blood sugar response to anoxia, and FRIEDMAN *et al.* (1956) reported a decline in neurohypophyseal function.

Histopathologic studies have supported the physiologic evidence of autonomic nervous system changes secondary to age. KUNTZ (1938) found a progressive change in dendrites, a reduction in chromidial substance, and a deposition of melanotic pigment in autonomic ganglion cells associated with age. The deposition of pigment, felt by CRITCHLEY (1942) to be the most important change, was least obvious in the hypothalamus.

Recently, however, ANDREW (1956) in an extensive study of structural alterations with ageing, has stated that the nuclei of the hypothalamus show perhaps the most impressive and striking incidence of specificity of age-change seen anywhere in the nervous system. He found an increase in cellular size to giant cell proportions, 8–10 times as large as comparable cells in younger individuals. There were multinucleate cells. The nucleolus showed multiple divisions and there was an increase in basophilic properties. These changes were felt to be a defensive reaction to ageing resulting in a disturbance of cellular functions (VOGT and VOGT, 1946).

To investigate further the physiologic changes due to ageing in hypothalamic reactivity, the present study was undertaken. An incentive to this study was the report of FUNKENSTEIN *et al.* (1949) that mental patients show a difference in the reactivity to mecholyl when compared with normals. There was no apparent control for age in their study. In view of the above evidence presented for a declining

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function in the autonomic nervous system with age, and in view of the recent work from this laboratory showing that the mecholyl test is a measure of the sympathetic and the noradrenaline-induced pulse slowing reflex a measure of the parasympathetic reactivity of the hypothalamus, it was felt the present study would be useful from two standpoints:

(1) To contribute to our knowledge of the declining reactivity of both divisions of the autonomic nervous system with increasing age.

(2) To verify the evidence presented that there is a difference in the autonomic nervous system reactivity between control subjects and mentally ill patients; and, if present, to determine what effect age has on this difference.

Evidence that Mecholyl and Noradrenaline Tests are a Measure of Hypothalamic Reactivity

Mecholyl. Earlier work (GELLHORN, 1957; GELLHORN *et al.*, 1956; GELLHORN and REDGATE, 1955; REDGATE and GELLHORN, 1955) on cats showed that an increase in the excitability of the sympathetic division of the hypothalamus caused a lessening in the hypotensive action of mecholyl and a tendency to an overshooting of the blood pressure following the hypotensive phase. Conversely, a diminution of sympathetic hypothalamic excitability was accompanied by an aggravation of the hypotensive action of mecholyl and a lessening or disappearance of the secondary hypertensive phase (overshooting). These results were obtained regardless of whether the alterations in hypothalamic reactivity were produced by the intrahypothalamic injection of drugs, electrical stimulation of the hypothalamus, or lesions confined to the posterior hypothalamus. Lesions in the thalamus, on the contary, failed to influence the mecholyl reaction.

Further experiments showed that mecholyl (and other hypotensive drugs) lead, via baroreceptor reflexes, to the excitation of the hypothalamus and the hypothalamic-cortical system (GELLHORN, 1957; NAKAO *et al.*, 1956).

Application of mecholyl in man leads to three distinct blood pressure curves (GELLHORN, 1953). Group I shows a slight hypotension followed by a prompt return to base line readings with subsequent overshooting of the previous resting levels. Group II shows a moderate hypotension with return to control levels within 8–10 min with no overshooting, and Group III shows a marked hypotension with delayed return to previous base line levels. In many cases, up to 25 min of continuous hypotension was observed in this group. In the light of experimental studies mentioned above, Group I was felt to represent increased, Group II normal, and Group III decreased posterior (sympathetic) hypothalamic reactivity.

Noradrenaline. In collaboration with REDGATE and NAKAO, GELLHORN (GELLHORN *et al.*, 1956; REDGATE and GELLHORN, 1956) showed that the cardiac slowing induced by the blood pressure rise from the injection of noradrenaline depends on the state of anterior (parasympathetic) hypothalamic reactivity.

Injection of pentothal and high-frequency coagulation of this structure greatly reduced the cardiac slowing seen under control conditions. Furthermore, after coagulation or pentothal injection, this slowing was reduced despite blood pressure responses in excess of the control conditions. In the case of pentothal, these effects were reversible.

EXPERIMENTAL PROCEDURE

Mecholyl test. The subject rested in bed until his blood pressure reached a base line which was 5 consecutive readings, 1 min apart, within 6 mm Hg. This period varied from 30 min to 2 hr. Ten milligrammes of mecholyl were injected intramuscularly. The blood pressure was then taken every

30 sec for 2 min, and every minute thereafter, for a total of 15 min. The test was not given to individuals with a past history of asthma, severe allergic reactions, or cardiac pathology.

The blood pressure record obtained from the injection of mecholyl was graphed on millimetre graph paper. The *Y* axis represented the blood pressure, with 2 cm equal to 10 mm Hg. The *X* axis represented time, with 1 cm equal to 1 min. By use of a Dietzgen polar planimeter, the area under the blood pressure curve was calculated. This method permitted exact limits to be set for each of the three groups. If the rebound of the blood pressure over its previous base line was greater than +100 vernier units (1 in.^{2}), the record was placed in Group I. If the area of the curve under the base line was between 550 to +99 vernier units (−5·5 to +0·99 in.^{2}) and if it was not followed by a hypertensive phase characteristic of Group I, the record was placed in Group II. Records showing a hypotensive area greater than −550 vernier units were placed in Group III. The range of measurements was from −1887 to +741 vernier units (18·87 to 7·41 in.^{2}).

Noradrenaline test. The same precautions were exercised as in the mecholyl test. The pulse rate was also determined. After it had remained constant within 6 beats/min for 5 min, noradrenaline (0·005 mg) was injected intravenously. The blood pressure and pulse rate were determined every 30 sec for 2 min, and every minute thereafter, for a total of 5 min. Pulse rates were derived from determination of time lapse for 15 pulse beats.

The index of parasympathetic reactivity was expressed as a ratio*—the amount of pulse rate slowing divided by the blood pressure rise. The larger the ratio, the greater the parasympathetic reactivity.

The control group totalled 111 persons. They had no past history of mental hospitalization nor of any type of mental illness. They were not under any undue emotional strain at the time of their test. Included in this group were college students, professional, working and retired people.

The experimental group numbered 241 mental hospital patients. The respective diagnostic categories are listed in Table 1.

Tests were done in both the acute and chronic wards at St. Peter State Hospital and Hastings State Hospital. Tests were also performed at the Minneapolis General and University of Minnesota Hospitals' psychiatric wards. Patients tested were not on somatic or psychotherapy. No correction was made for length of illness, although the age groupings closely paralleled it. The means and standard deviations of each age group to be discussed are listed in Table 2. There was no apparent sex difference in either group in the reaction to the two tests.

TABLE 1.—DIAGNOSIS IN EXPERIMENTAL GROUP

Schizophrenia	128
Psychopathic personalities	37
Personality disorders (Pass.—Agg.—Juv. Delin.—Alcoholics)	16
Psychoneurosis	14
Manic depressive	5
Depression	5
Other	36
Total:	241

RESULTS

Mecholyl test. The results of the mecholyl test in the experimental and control groups are shown in Table 3. Both groups show a decline in group I (consisting of sympathetic hyperreactors) and an incline in group III (sympathetic hyporeactors) as the age of the sample increased. This effect of age on the sympathetic reactivity

* i.e. $\frac{\text{Mean of resting pulse rates minus minimal pulse rate}}{\text{Maximal systolic blood pressure minus mean of resting systolic blood pressures}}$

TABLE 2. MEAN (M) AND STANDARD DEVIATION (S.D.) OF CONTROL (C) AND EXPERIMENTAL (E) AGE GROUPS

Age 25 and less:	
CM—20·1 years	S.D.—2·1 (50 cases)
EM—19·8 years	S.D.—2·7 (39 cases)
Age 26–35:	
CM—29·3 years	S.D.—2·8 (24 cases)
EM—31·0 years	S.D.—2·9 (67 cases)
Age 36–45:	
CM—39·1 years	S.D.—2·8 (11 cases)
EM—40·6 years	S.D.—2·7 (63 cases)
Age 46 and over:	
CM—51·8 years	S.D.—4·4 (20 cases)
EM—53·7 years	S.D.—6·1 (71 cases)

TABLE 3. THE EFFECT OF AGE ON THE REACTIVITY TO MECHOLYL

Control group				Experimental group			
Age 25 and less:							
I	14	31%		I	20	56%	
II	31	69%		II	11	30%	
III	0			III	5	14%	
	45				36		
Age 26–35:							
I	4	18%		I	25	40%	
II	18	82%	$\chi^2 = 18{\cdot}19$	II	31	50%	$\chi^2 = 39{\cdot}30$
III	0			III	6	10%	
	22		$p < 0{\cdot}01$		62		$p < 0{\cdot}001$
Age 36–45:							
I	4	25%		I	24	37%	
II	11	69%		II	31	49%	
III	1	6%		III	9	14%	
	16				64		
Age 46 and over:							
I	1	5%		I	12	17%	
II	14	67%		II	27	36%	
III	6	28%		III	35	47%	
	21				74		

measured by the mecholyl test was highly significant ($p < 0{\cdot}01$ in the normal, and $< 0{\cdot}001$ in the experimental group).

The effect of age on the action of mecholyl in the largest clinical group, the schizophrenes, totalling 124, is shown in Table 4. Here again the mecholyl Group I showed a progressive decrease with increasing age. The changes were likewise highly significant ($x^2 = 20{\cdot}72$; $p < 0{\cdot}001$).

An inspection of the data shown in Tables 3 and 4 discloses characteristic differences between the control and experimental groups, although the effect of age applies to both groups. It is noted that group II represents the majority at all ages of the control cases, but only 30–50 per cent of the experimental group. Assuming that group II represents the "norm", it appears that deviations from this norm are more common in the experimental than in the control groups at all age levels. The statistical analysis showed that the control and experimental groups differ significantly regardless of age ($\chi^2 = 23{\cdot}62$ with 6 degrees of freedom, $p = 0{\cdot}001$).

TABLE 4. THE EFFECT OF AGE ON THE REACTIVITY TO MECHOLYL IN SCHIZOPHRENES

Age 25 and less			26–45			46 and over			Total	
I	7	54%	I	25	37%	I	8	18%	40	$\chi^2 = 20{\cdot}72$
II	2	15%	II	37	54%	II	16	37%	55	$p < 0{\cdot}001$
III	4	31%	III	6	9%	III	19	45%	29	
	13			68			43		124	

With increasing age there was not only a shift from group I to group III but also an increase of the mean blood pressure. In order to eliminate the influence of an alteration of the blood pressure level on the reactivity to mecholyl the results were retabulated excluding all records with resting blood pressures of 150 mm Hg and above. Tables 5 and 6 show that the ageing process causes a shift from group I to group III in the control and experimental groups despite the exclusion of hypertensives and the virtual absence of a significant rise of the blood pressure with increasing age. The statistical analysis shows that the changes due to age are significant in these control and experimental groups ($p < 0{\cdot}01$). A comparison of these groups at similar age levels leads to the following results:

Significance of differences between the control and experimental groups.

(I) Age 25 and less	$\chi^2 = 16{\cdot}6$	$p < 0{\cdot}01$
(II) Age 26 to 45	$\chi^2 = 8{\cdot}9$	$p < 0{\cdot}015$
(III) Age 46 and more	$\chi^2 = 2{\cdot}5$	$p < 0{\cdot}3$

The progressive increase in the number of sympathetic hyporeactors with increasing age tends to minimize or eliminate the significant differences in sympathetic reactivity which exist between the control and experimental groups at the age of 25 years and less.

The mecholyl test was found to be highly reliable. It was repeated in 11 cases, the interval between tests being a week, and only one response was not repeatable

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TABLE 5. ANALYSIS OF MECHOLYL GROUPINGS IN INDIVIDUALS WITH BLOOD PRESSURES 149 MM/HG AND BELOW

Normals

Age 25 and less:

Mean of blood pressures		117·5 mm/Hg
Standard deviation		10·90
Mecholyl grouping:		
Number		Percentage
I	14	31
II	31	69
III	0	
	45	

Age 26–45:

Mean of blood pressures		117·5 mm/Hg
Standard deviation		8·70
Mecholyl groupings:		
Number		Percentage
I	9	24
II	28	74
III	1	2
	38	

$x^2 = 24{\cdot}4$
$p < 0{\cdot}01$

Age 46 and above:

Mean of blood pressures		122·9 mm/Hg
Standard deviation		11·25
Mecholyl groupings:		
Number		Percentage
I	1	5
II	13	69
III	5	26
	19	

(5 were group I, 3 in group II, and 3 in group III). These results are in agreement with the work of WECKOWICZ (1956).

Noradrenaline test. The noradrenaline test was repeated in thirteen controls. The correlation between repeated tests was 0·80, and by the use of the t test, was found to be significant ($p < 0{\cdot}001$).

The means and standard deviations of the index of parasympathetic reactivity (P.I.) as it relates to increasing age are recorded in Table 7. There was a gradual

TABLE 6.—ANALYSIS OF MECHOLYL GROUPINGS IN INDIVIDUALS WITH BLOOD PRESSURES 149 MM/HG AND BELOW

Experimental Group

Age 25 and less:

Mean of blood pressures 122·5 mm/Hg
Standard deviation 10·05
Mecholyl grouping:

	Number	Percentage
I	20	57
II	10	29
III	5	14
	35	

Age 26–45:

Mean of blood pressures 123·05 mm/Hg
Standard deviation 11·7
Mecholyl grouping:

	Number	Percentage
I	47	40
II	57	48
III	14	12
	118	

Age 46 and above:

Mean of blood pressures 130·95 mm/Hg
Standard deviation 11·95
Mecholyl grouping:

	Number	Percentage
I	8	14
II	27	49
III	20	37
	55	

$x^2 = 25{\cdot}96$
$p < 0{\cdot}01$

decrease in this mean with increasing age. This gradual reduction was present in both groups. The difference between means of the youngest and oldest age groups, in both samples, was found to be statistically significant ($p < 0{\cdot}001$, using the t test). There appeared to be little difference between the control and experimental groups when matched for age. Table 7, therefore, demonstrates a progressive decrease in the reactivity of the parasympathetic nervous system as the age of the subjects tested increased.

TABLE 7.—MEAN (M) AND STANDARD DEVIATION (S.D.) OF THE INDEX OF PARASYMPATHETIC REACTIVITY IN CONTROL (C) AND EXPERIMENTAL (E) GROUPS AS RELATED TO AGE

Age 25 and less:		
	CM—0·68	S.D.—0·37
	EM—0·68	S.D.—0·20
Age 26–35:		
	CM—0·64	S.D.—0·35
	EM—0·61	S.D.—0·39
Age 36–45:		
	CM—0·41	S.D.—0·30
	EM—0·50	S.D.—0·40
Age 46 and over:		
	CM—0·32	S.D.—0·29
	EM—0·32	S.D.—0·30

Figs. 1 and 2 show on the basis of the raw data obtained in the control and experimental groups the influence of age on the parasympathetic reflex excitability. The reciprocal of the index of parasympathetic reactivity chosen as the abscissa varied between 0 and more than 5. As the tables indicate, age causes a marked shift in the percentage of the subjects toward the higher quotients, i.e. to a lessened parasympathetic responsiveness. This result was obtained regardless of whether the change in parasympathetic reactivity was determined on the basis of the absolute changes of the blood pressure and pulse rate, as in Figs. 1 and 2, or on the relative changes expressed in percentages of the resting blood pressures and heart rates.*

In the tabulation of the data it was noted that in the control group the parasympathetic reactivity increased with increasing resting pulse rates. This suggested that the noradrenaline-induced parasympathetic reactivity was determined not only by the age level but also by the resting pulse rate.

This phenomenon is illustrated in Figs. 3 and 4, in which the influence of the resting pulse rate on the reciprocal of the parasympathetic index is shown for the control and experimental groups. It is seen that with increasing pulse rate the percentage of subjects with lower quotients increases markedly in the control group whereas the experimental group fails to show this effect. Here again these results were obtained not only when the blood pressure and pulse rate changes were plotted on a percentage basis, as in Figs. 3 and 4, but also when plotted on the basis of the absolute changes in blood pressure and pulse rate. In order to investigate this relation further, the data in the control groups were analysed statistically for the age groups *25 and less* and *46 and older*. Each group showed a correlation of 0·58 between the index of parasympathetic reactivity and the resting pulse rate which was significant for the young and old groups ($p < 0{\cdot}001$, $p < 0{\cdot}01$ respectively), confirming our impression that the P.I. increased with increased pulse rate.

Figure 5a demonstrates for the control group the lines that best fit the data (calculated by least-squares method) for each age group. Here once more the effects of age are easily noted, namely a loss in reactivity in later life. In addition, the figure shows that in the control group the P.I. increases with increasing resting pulse rate. If it

* Note that in Figs. 1–4, the abscissa is reciprocally related to P.I.

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may be assumed that the resting pulse rate is primarily determined by the vagal tone the results indicate that the noradrenaline-induced reflex slowing of the heart rate decreases with increasing tone of the vagal nerve.

Fig. 5b shows similar calculations applied to the corresponding data of the experimental group. There appears to be very little correlation between resting

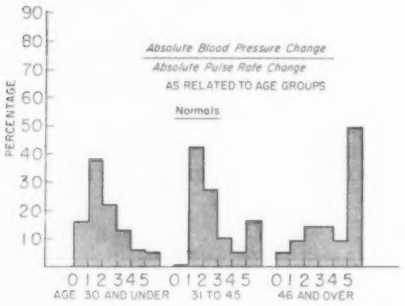

FIG. 1.—The influence of age on the reciprocal of the parasympathetic reactivity (control group). The shift to the right seen in Figs. 1 and 2 indicates a decrease in parasympathetic reactivity with increasing age.

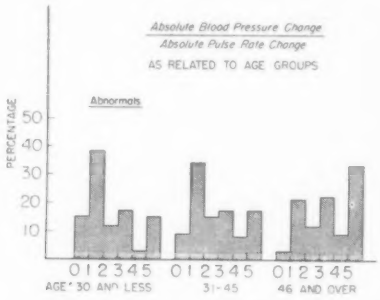

FIG. 2.—Same as Fig. 1 for the neuropsychiatric group.

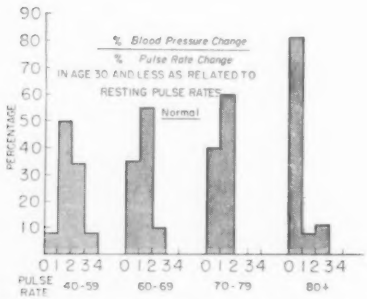

FIG. 3.—The influence of the resting pulse rate on the reciprocal of the parasympathetic reactivity (control group). The shift to the left indicates an increase in parasympathetic reactivity with increasing pulse rate.

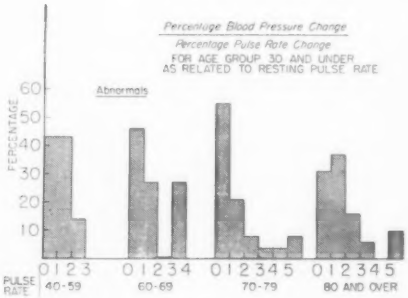

FIG. 4.—Same as Fig. 3. for the neuropsychiatric group. No significant change in relation to the resting pulse rate.

pulse rates and the parasympathetic reactivity in mental patients. At age *25 and less*, the correlation coefficient was 0·12 and for age *46 and over*, 0·25.* The difference in P.I. between the two age groups is, however, quite evident. It is also apparent that when the graphs for the control and experimental groups are compared, the aging process, among other factors, tends to bring the two groups closer in their distribution.

The equations for the graphs shown in Fig. 5 enable one to predict the expected index of parasympathetic reactivity for a given resting pulse rate. This *expected* index was calculated for each subject in both the experimental and control groups, using the equation derived from the data of the control group of age *25 and less*.

* Similar differences between the control and experimental groups were disclosed by the χ^2 test.

The *calculated* index was next subtracted from the *observed* index obtained by experimental means. It was found that in the control group of age *25 and less*, the mean of the difference (*observed* minus *calculated*) was 0 and the standard deviation was 0·3. On this basis it was assumed that if the *observed* minus *calculated* difference was greater than 0·3, it represented an increased parasympathetic reactivity: such cases

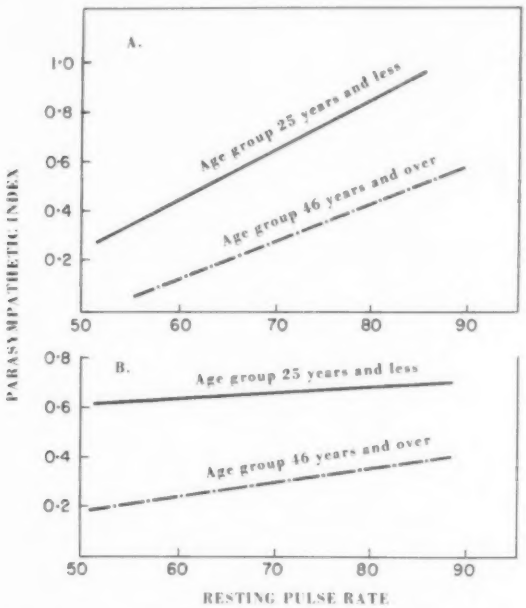

FIG. 5.—Parasympathetic Index as related to age and pulse rates in the control group (A) and the experimental group (B).

were placed in group I. Group II was thought to be normal in reactivity and its limits were placed at 0, plus or minus 0·3. If the difference was of a greater negative value than 0·3, it seemed to represent a decreased parasympathetic reactivity. These cases were designated as group III. Table 8 represents the frequency distribution of the groups so defined as they relate to age. It is seen, much as in the sympathetic reactivity, that there is a decline in group I (parasympathetic hyperreactors) and an incline in group III (parasympathetic hyporeactors) as the mean age of the population increases. These changes are statistically significant ($p < 0{\cdot}01$) for the control and experimental groups. The table further indicates that with increasing age the differences in the distribution of the parasympathetic index are diminished and are no longer significant at the age of 46 and older. At the youngest age group, however, differences are significant and show that deviations from the norm are more common in the experimental than in the control group.

EVALUATION OF AUTONOMIC BALANCE

In Table 9 an attempt was made to utilize our data based on the mecholyl and noradrenaline tests for the evaluation of the autonomic balance in the control and experimental groups. It was determined how often a sympathetic hyperreactivity (mecholyl group I) was combined with a parasympathetic hyporeactivity. This form

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TABLE 8.—DISTRIBUTION OF GROUPS I, II AND III AS MEASURED BY OBSERVED PARASYMPATHETIC REACTIVITY MINUS CALCULATED PARASYMPATHETIC REACTIVITY.*

Control			Experimental			Significance of difference between control and experimental groups
Age 25 and less:						
I	9	18%	I	10	25%	$x^2 = 16{\cdot}3$
II	35	70%	II	12	31%	$p < 0{\cdot}01$
III	6	12%	III	17	44%	
	50			39		
Age 26–35:						
I	5	21%	I	9	14%	$x^2 = 6{\cdot}61$
II	17	71%	II	37	55%	$p = 0{\cdot}05$
III	2	8%	III	21	31%	
	24			67		
Age 36–45:						
I	1	9%	I	5	8%	
II	6	55%	II	31	49%	
III	4	36%	III	27	43%	
	11			63		
Age 46 and over:						
I	0	0%	I	3	4%	$x^2 = 1{\cdot}6$
II	7	35%	II	26	37%	$p = 0{\cdot}5$
III	13	65%	III	42	59%	
	20			71		

Significance of changes with age

$x^2 = 28{\cdot}54$	$x^2 = 20{\cdot}66$
$p < 0{\cdot}01$	$p < 0{\cdot}01$

* Persons showing a difference exceeding plus one standard deviation were placed in Group I; persons in whom the difference was more negative than minus one standard deviation were placed in Group III and those with less positive or negative differences were placed in Group II.

of imbalance occurred more often in the experimental than in the control group. The opposite type of imbalance, sympathetic hyporeactivity (mecholyl group III) in combination with parasympathetic hyperreactivity did not occur in the control and only to a slight extent in the experimental groups. The table also shows that these results apply to the largest clinical group, the schizophrenes. As previously noted, the age factor tends to obliterate the autonomic differences existing between the control and experimental groups. This holds true for the frequency of autonomic imbalances.

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Finally, in section C of Table 9 the frequency of all forms of autonomic imbalance as disclosed by our tests was determined. It was assumed that the combination of mecholyl group I with parasympathetic index I represented a balance between the two divisions of the autonomic system at the hyperreactive level, whereas mecholyl group II with parasympathetic index II represented the autonomic balance at the intermediate, and mecholyl group III with parasympathetic index III at the hyporeactive levels. The diminution in the frequency of occurrence of autonomic hyperreactivity and the increasing dominance of autonomic hyporeactivity with increasing age

TABLE 9.—AUTONOMIC IMBALANCE IN NORMAL AND EXPERIMENTAL GROUPS

	Age	Normals	Abnormals	Schizophrenes
A. Mecholyl Group I combined with parasympathetic Index III	25 and less	3%	26%	38%
	26–35	0%	10%	11%
	36–45	9%	6%	11%
	46 and over	5%	11%	11%
B. Mecholyl Group III combined with parasympathetic Index I	25 and less	0%	3%	8%
	26–35	0%	3%	0%
	36–45	0%	2%	3%
	46 and over	0%	1%	0%
C. All forms of autonomic imbalance	35 and less	45%	71%	70%
	36–45	46%	69%	76%
	46 and over	35%	51%	52%

suggest that the autonomic balance may be maintained at different levels of activity. All combinations in which the mecholyl group and the parasympathetic index showed different numbers were considered to be cases of imbalance. Table 9 C shows again that the autonomic imbalance appears more frequently in the experimental than in the control groups and that this difference declines with increasing age.

DISCUSSION

The work described in this paper establishes the fact that age causes a statistically significant increase in the hypotensive action of mecholyl. It is suggested that this shift in the action of mecholyl indicates a decrease in sympathetic (hypothalamic) excitability. This interpretation is supported by the physiological studies cited in the introduction.

The change in the action of mecholyl on the blood pressure with increasing age might theoretically be due to a change in the action of mecholyl on the blood vessels and/or on sympathetic centres in the brain stem in varying proportions. The interpretation that the changes disclosed by the mecholyl test as the result of aging are due to the action of age on central autonomic structures and particularly on the hypothalamus is given preference for the following reasons:

(1) The hypotensive action of mecholyl increases with increasing age although hypertensive cases were eliminated and the mean blood pressure remained practically constant. It is improbable that the state and reactivity of peripheral structures such as the blood vessels are greatly altered under these conditions.

(2) Numerous investigations have shown that alterations of the internal environment as in

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anoxia, hypoglycaemia and other conditions (GELLHORN, 1943) affect primarily the central and not the peripheral structures. Within the central nervous system the cortical and diencephalic structures are more sensitive than the medulla oblongata and spinal cord (HIMWICH, 1951). This principle of a central gradient of reactivity is involved in the mecholyl test. It was shown that the action of mecholyl on the blood pressure depends primarily on the baroreceptor reflexes (NAKAO *et al.*, 1956). These reflexes are still present after the elimination of the diencephalon, but their sensitivity is lessened. Consequently, the hypotensive action of mecholyl is increased and the secondary hypertensive phase is eliminated as the sensitivity of the sympathetic division of the hypothalamus is lessened. It is believed that such a loss in sensitivity is primarily involved in the alterations of the mecholyl test resulting from age.

(3) FUNKENSTEIN *et al.* (1949) observed that changes in the psychological state, regardless of whether they occur spontaneously, as the result of psychotherapy, or following electroshock treatment, are associated with changes in the mecholyl test. These data seem to illustrate for the human the great influence of the emotional state on the mecholyl test. In the light of our previously cited experiments on cats this clinical work presents additional evidence in favour of our assumption that the mecholyl test measures primarily the excitability of the sympathetic division of the hypothalamus in the human.

The noradrenaline-induced slowing of the heart rate was employed as a test for the excitability of the parasympathetic system. This reflex depends on the excitability of the centres of the parasympathetic system. Here again it was found that the magnitude of this reflex in response to a given noradrenaline dose depends on the excitability of the hypothalamus—in this instance of the anterior (parasympathetic) division, since the functional elimination of this division diminishes the reflex slowing (GELLHORN *et al.*, 1956; REDGATE and GELLHORN, 1956). The diminution of this reflex observed with increasing age in control and experimental groups is interpreted as being due to a diminishing reactivity of the parasympathetic hypothalamus. Theoretically it is conceivable that the reactivity of the peripheral blood vessels to noradrenaline may change with increasing age. Even if this were the case it would not be of any consequence for our interpretation of the noradrenaline test as an indicator of central (hypothalamic) parasympathetic reactivity, since the parasympathetic index of our experiments is based on the *ratio* of the pulse slowing to the rise of the blood pressure!*

The mecholyl and noradrenaline tests have further shown that characteristic differences between the control and experimental groups exist. Sympathetic hyperreactors and hyporeactors (indicated by mecholyl group I and III respectively) are more frequent in neuropsychiatric patients than in the control group. These differences are most marked in the age group of 25 and less and become progressively less as the age increases. It has been suggested by one of us (GELLHORN, 1953, 1956, 1957) that the type of autonomic (hypothalamic) disorder disclosed by mecholyl test should be taken into consideration for the selection of the form of therapy to be applied in a particular patient. Since the age factor tends to obscure and, eventually, to obliterate the differences in autonomic reactivity between the control and experimental groups, it appears best to apply the above-mentioned therapeutic considerations primarily to patients of the low age group.†

* The mean rise of the blood pressure on injection of noradrenaline at the age groups of 25 and less, 26–45, and 46 and older was (in mm Hg) for the control groups 20, 20 and 31 and for the experimental groups 19, 21, and 21 respectively. Since the P.I. decreased in both groups with increasing age and significant differences in the P.I. existed between the young and the older groups the changes in peripheral responsiveness seem to be unrelated to changes in the parasympathetic index produced by age.

† Since age tends to increase the percentage of the sympathetic hyporeactors, the persistence of a sympathetic hyperreactivity in the older age group appears to be a trustworthy indication of an autonomic disturbance.

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Although the parasympathetic reactivity showed wide individual differences, it was possible, on the basis of certain statistical procedures (see Table 8) to divide the data into groups of parasympathetic hyperreactors and hyporeactors and an intermediate group, as was done previously with the mecholyl tests. On this basis it could be shown that parasympathetic hyperreactors and hyporeactors are more common in the experimental than in the control groups and that autonomic imbalances at various levels of sympathetic reactivity occur more frequently in neuropsychiatric disorders than in the control group.

SUMMARY

The results of the injection of mecholyl on the blood pressure and of the injection of noradrenaline on the reflex slowing of the heart rate on a large number of "normals" and neuropsychiatric patients at varying age groups are reported. On the basis of the studies on experimental animals with these drugs the mecholyl test is considered to be an indicator of the sympathetic, and the noradrenaline test an indicator of parasympathetic reactivity at the hypothalamic level. It is found that the sympathetic reactivity decreases with increasing age. This statement holds for the normal and experimental groups and, among the latter, for the largest clinical division to which these tests were applied, the schizophrenes. The decline of the sympathetic reactivity with increasing age is not due to the increasing blood pressure since it persists after elimination of the cases with hypertension. The parasympathetic reactivity disclosed by the noradrenaline test shows likewise a decline with increasing age in the control and experimental groups.

A comparison of the control and experimental groups at similar age levels shows that sympathetic and parasympathetic hyperreactors and hyporeactors are more frequent in the experimental than in the control group. These differences become less with increasing age. The parasympathetic reactivity increases with increasing pulse rate at rest in the control group but not in the experimental group. Finally, autonomic imbalances at different levels of sympathetic reactivity occur more frequently in the experimental than in the control group.

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A PSYCHIATRIC INVESTIGATION OF THE DELAY FACTOR IN PATIENT TO DOCTOR PRESENTATION IN CANCER*

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INTRODUCTION

IN CANCER, more than any other disease process, delay in seeking treatment has far-reaching effects. It is accepted that the longer the patient delays, the less are his chances of recovery. It is considered, therefore, that an investigation into the factors relating to the early or late presentation to medical care is of great importance both to individuals and to the population as a whole. From such an investigation, information may emerge whereby measures may be designed to assist the early appearance of patients in the treatment situation. In the field of cancer, the words "early" and "late" have particular meanings related to the clinical or pathological extent of the lesion. An early case is one in which the cancer is accepted clinically or pathologically as not being advanced in extent. Therefore, this investigation of delay factors has come under the two headings: (a) temporal evidence and (b) pathological evidence.

In the series of one hundred patients, temporal delay is defined as a period of three months or over between the onset or discovery of symptoms and the first presentation to the doctor. This is in accordance with the definition of delay originally given by PACK and GALLO (1938).

BASIC HYPOTHESES

(1) It was hypothesized that the site of the lesion and the initial symptomatology influence to some extent the time at which the patient presents himself. For instance, it appears likely that a patient with a lesion on the body surface establishes an earlier contact with the doctor than a patient with a malignant growth of the body interior; and that discharge or bleeding from a nipple would be more likely to cause an earlier presentation to the doctor than increasing constipation or discharge from the female genital tract.

(2) It was hypothesized that early or late presentation is related to the personality of the individual concerned.

(3) The hypothesis was made that cancer education may, in some instances, be highly effective while in others it may foster delay, depending on personality factors involved in individual cases and also on the nature of the educational material. Huge sums of money are spent every year on bringing information on cancer to the general public. The effectiveness or otherwise of the various methods of cancer education must be investigated.

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Source of Clinical Material and Description of Research Procedure

(1) *Setting for the research*

The patients were drawn from the two largest English-speaking hospitals in Montreal. In ages they ranged from 24 to 81 years. There were 11 males in the series. The socio-economic status varied from patients who were existing on a meagre pension to representatives of high-salaried professional classes. The majority of the patients were either born in Canada or had been resident there many years. Recent immigrants were also present in the series investigated.

In this investigation cancer of the breast, the cervix, and the large bowel, which included cancer of the rectum, was studied. As mentioned before, these sites were chosen to ascertain differences, if any, in relation to site and symptomatology of the lesion. The breakdown of the 100 cases by diagnosis is shown in Table 1.

TABLE 1.—BREAKDOWN OF 100 CASES BY DIAGNOSIS

Diagnosis	Number of cases	
Carcinoma of breast		63
Carcinoma of cervix		18
Carcinoma of large bowel, including rectum	F:	8
	M:	11
Totals		100

(2) *Description of the research method*

The population can be defined as follows:

A non-selected group of patients of varying ethnic origin, educational standards, and intelligence levels was examined. Both urban and city domicile was represented in the group. They were of varied social and economic levels. The patients were included in the series because of two factors; one, the presence of a cancer in any of the defined sites, and the other, that they had placed themselves under treatment in hospital for the disease.

All patients in the series were investigated by a combination of non-directive and mildly directive interview techniques. Patients were examined at varying times in the history of the treatment schedule. Some were seen soon after admission to hospital, when it was possible to ascertain their attitude to entering hospital, and observe the psychological defence mechanisms mobilized to cope with the admission situation. Others were seen before and after operation, or before and after attending the X-ray department for radiation therapy. Care was taken to see that no patient was examined during any spell of severe physical discomfort or under the influence of excessive amounts of sedation. Each patient was seen for a minimum period of 2 hr, though many had second interviews.

To begin with, the patient was encouraged to speak about his illness and all that it meant to him. When information on this subject had been obtained in a non-directive manner, factual information was elicited regarding the patient's past medical history and general life situation. Finally, the interview situation became mildly directive to ascertain the reaction to cancer as a diagnosis and a disease, the contact with cancer education, the reaction to the treatment situation, and, of course, reasons for delay or non-delay.

Attention was paid throughout the interview not only to what the patient said, but also to how he said it, and to any clues to unconscious mental processes given by the patient's appearance and behaviour during the interview. In addition, an appraisal was made of obvious areas of conflict in his life

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situation and of the ego defence mechanisms normally used by him. From all the information available a formulation was made of each patient's personality.

Method of Handling Data and Definition of Terms Employed

(A) *Breakdown of cases*

Using the definition of delay given above, the 100 cases were divided into delay and non-delay groups. A factorial analysis was carried out in each case regarding the relevance of the disease, the personality of the patient, and of the effect of cancer education for delay or non-delay.

(i) *Factors connected with the disease.* Factors considered were site of cancer, early symptoms of disease, and similarity of symptoms due to some other disease process. The site of the cancer, as, for example, cancer of the cervix, might be an important factor in causing delay if the patient looked upon the bleeding and discharge as indicating an excessive menstrual flow or due to the approaching menopause.

(ii) *Factors connected with personality.* Emotional maladjustment in general, and anxiety and depressive features in particular, were examined and compared in the two groups.

(iii) *Factors connected with cancer education.* In order to assess how effective cancer education campaigns had been in reaching this particular population, and whether this exposure to cancer education reduced or prolonged the period of delay, extensive study was made of the verbatim reports of patients in the two groups concerning their attitude to cancer education and the knowledge they had obtained from such educational material.

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(B) *Definition of terms employed in discussion*

(i) *Education.* Many of the patients in this series belonged to a generation in which education was voluntary, and accordingly the educational standards reached were often minimal. It proved difficult to arrive at a definite educational standard which could be used as a common denominator for the whole group. Grade 8 of the Canadian Educational System has been taken as the average educational standard for assessing the patients on an 8-plus or -minus scale.

(ii) *Intelligence.* The following criteria were taken into account in appraising the intelligence grading: (1) educational standard reached; (2) occupational history of the patient; (3) vocabulary used and conceptualizations introduced at interview.

On the basis of these criteria the patients were divided into three groups of intelligence: average (A); less than average (A−); and above average (A+).

(iii) *Previous contact with cancer.* More than one form of cancer contact can be recognized. The contact can be close, as in the immediate family or friends, or distant, as in acquaintances, and yet the effect of the latter may be as important as that of the former. A sub-division of contact was made into: (a) family of origin; (b) family of orientation; (c) experience of cancer outside the family constellation.

(iv) *Cancer education.* In this investigation, exposure to cancer education has been defined as contact with any medium which has produced, or would tend to produce, increased knowledge of the disease. This includes the spoken word of lectures and addresses; the written word of papers, pamphlets, booklets, newspapers and advertising generally; films and television.

Furthermore, since everybody can be exposed to cancer education if he so desires, it is important to find the answers to several questions.

(1) Are rural populations which, on the whole, are less exposed to cancer education, more likely to delay than others?

(2) The literature on Delay contains references to "ignorance" (AITKEN-SWAN and PATERSON, 1955; BATES and ARIEL, 1948; HARMS *et al.*, 1943; PATERSON, 1955; SCHEFFEY, 1953; YOUNGMAN, 1947) as a reason for the cancer patient delaying. Is this alleged ignorance a real entity or is it due to resistance on the part of the patient, which is allied to the defence mechanisms of repression, suppression, and negation (denial)?

(3) Finally, is there any particular form of cancer education which brings the patient to the doctor and any form which keeps him away?

(v) *Contact of patient with doctor.* Whether delay has or has not occurred, there comes a time when the individual decides that action is indicated and consultation with the physician occurs. It was felt that investigation of the reasons for presenting to the doctor would reveal interesting information, and therefore a triplicate division was made into the headings: (a) lesion, (b) symptoms, and (c) other reasons for initial presentation.

(vi) *Defence mechanism.* The habitual defence mechanisms used by the patient were examined, and particularly those defences brought into play by the patient in dealing with the appearance of the initial cancer symptoms and the anxiety thereby produced.

(vii) *Knowledge of the diagnosis.* Knowledge of the diagnosis may be conscious or pre-conscious.

FENICHEL (1945) points out that in small children a "wish-fulfilling denial of unpleasant realities" is common, and that in the adult, these tendencies to deny tend to remain operative. An expression of the infantile "denial in phantasy" is seen in the adult (and this particularly refers to many patients with cancer) who, knowing an unpleasant truth, indulges in daydreams or thoughts that deny the truth. The term "repression" refers to a defence of the ego against instinctual danger and affect. Therefore it is conceivable that an individual acknowledges the diagnosis but represses the affect of anxiety which it arouses. On the other hand, the ego may be protected against painful anxiety by projecting and displacing the source of anxiety. The individual who is partly aware of having a cancer and all that this means, may deal with his anxiety by expressing a fear of doctors and hospitals, and may thus hope by avoiding doctors and hospitals to avoid the fatal result of the disease.

(viii) *Loss of an important person.* Recent studies by LESHAN and WORTHINGTON (1955, 1956) discuss the importance of "Loss of an important relationship before the diagnosis of tumour." An individual may express most of his psychic energy through such an important person. It was postulated, for the purpose of this study, that the loss of such a person would be a factor tending to produce depression, an increased state of passivity, and therefore delay. The loss of an important person was therefore investigated under the headings: (a) loss of a parent, (b) loss of spouse, (c) loss of another important person.

FINDINGS

Out of the 100 patients studied, 69 had delayed, as against 31 in the non-delay group.

Stated Reasons for Delay

Each patient, whether the delay period was long or short, was asked why he delayed. In addition, whether the patients delayed or not, an enquiry was made regarding the reasons for delay attributed to others. The reasons found for the delay by the investigator did not always coincide with those stated by the patients. Table 2 shows the reasons given by 100 patients in the discussion of delay, either by themselves or by others. More than one reason for delay may be given by the same patient.

Item 1: *Symptoms not considered serious enough.* Forty-three-point-five per cent of patients in the delay group gave this reason, a much higher percentage than in the non-delay group. Typical of many others is the patient aged 47 years who had a cancer of the cervix and delayed seeking medical assistance for 7 months. The presenting symptom was vaginal haemorrhage which appeared intermittently. "I had

TABLE 2.—STATED REASONS FOR DELAY BY PATIENTS IN THE DELAY AND NON-DELAY GROUPS FOR SELF AND OTHERS

	Delay (69 cases)				Non-delay (31 cases)			
	Self	%	Others	%	Self	%	Others	%
(1) Symptoms not considered serious enough	30	43·5	5	7·2	4	12·9	5	16·1
(2) Misidentification with previous illness	8	11·6	—	—	1	3·2	1	3·2
(3) No pain	12	17·4	1	1·4	4	12·9	2	6·4
(4) No previous history of illness	4	5·8	3	4·3	1	3·2	2	6·4
(5) The belief that nothing can be done for them	2	2·9	3	4·3	—	—	4	12·9
(6) Symptoms regarded as normal	3	4·3	—	—	—	—	—	—
(7) Previously frightening experiences with patients who died of cancer	4	5·8*	—	—	—	—	—	—
(8) Age (i.e. "too old to bother" seeking treatment)	4	5·8	—	—	1	3·2	—	—
(9) Could not leave their work	4	5·8	1	1·4	—	—	1	3·2
(10) Expense of treatment	19	27·5	16	23·2	—	—	14	45·2
(11) No time	3	4·3	—	—	—	—	1	3·2
(12) Embarrassed at being examined by a doctor	7	10·1	5	7·2	1	3·2	2	6·4
(13) Fear of:								
(a) doctors	17	24·6	17	24·6	1	3·2	17	54·8
(b) hospitals	2	2·9	5	7·2	1	3·2	6	19·4
(c) operations	9	13·0	10	14·5	1	3·2	2	6·4
(14) Fear of dying	2	2·9	1	1·4	—	—	1	3·2
(15) Fear of what they will be told	8	11·6	8	11·6	1	3·2	12	38·7
(16) Thinking of others before themselves	7	10·1	1	4·2	2	6·4	1	3·2
(17) Miscellaneous	2	2·8	—	—	—	—	—	—

* Analysis of the clinical interviews showed that in fact a much higher percentage of the delay group had previous experience of cancer. The apparent discrepancy is due to the fact that many patients did not explicitly state that their delay was due to previous contact with cancer.

thought it was the change of life with very bad headaches. It didn't worry me at all. It wasn't like a lot of bleeding. It wasn't much."

Item 2: *Misidentification with previous illness.* This reason for delay was given by 11·6 per cent of delaying patients in contrast to 3·2 per cent of the non-delaying patients.

For example, a female patient aged 57 years had a history of repeated episodes of bleeding piles of long standing. The patient delayed for a period of 7 months. She described her attitude to the rectal bleeding, which was the initial symptom of her rectal carcinoma, as follows:

"In winter time the house was cold. I was always freezing and I started having bleeding piles. I didn't pay much attention until one day I notices the bowl full of blood. I thought I had bleeding piles. I have had piles, you know."

Item 3: *No pain.* The fact that this reason is given practically equally by both groups reflects the importance of pain in the minds of the public. Pain is equated as indicating something seriously wrong, and therefore "no pain" is the equivalent of being "not serious."

Item 10: *Expense of treatment.* In this study certainly, the subject of expense of medical treatment recurred frequently in the case histories, and it must be accepted as a moderately realistic factor in leading to delay, though individual personality dynamics cannot be ignored.

Item 13A: *Fear of hospitals.* This fear, expressed by patients in both the delay and non-delay groups, is worthy of mention. Fear of doctors has been interpreted above as a projective defence against anxiety aroused by the disease. Fear of hospitals is based on a childlike belief in the security of the home environment; the hospital atmosphere, by contrast, is regarded as strange, disquieting, and potentially dangerous.

Found Reasons for Delay

These will be discussed under the following headings: the physician, the disease, the patient, and cancer education.

(A) *Factors connected with the physician*

In contrast to observations made by others, in only 4 patients in the series was the delay period apparently either completely or partially due to the physician (LEACH and ROBBINS, 1947; PATERSON, 1955; PACK and GALLO, 1938; ROBBINS *et al.*, 1950, 1953).

(B) *Factors connected with the disease*

Table 3 presents the breakdown of the 100 patients according to diagnosis.

(i) *Minor symptoms overlooked.* Fifty-point-seven per cent of patients in the delay group had overlooked minor symptoms, such as vague aches and pains, occasional discharge or increasing constipation, as against 9·7 per cent of the patients in the non-delay group.* As a demonstration, one patient with a cancer of the rectum delayed 6 months and regarded the first appearance of blood in the bowel movement as simply due to piles, a condition which he had never previously had.

(ii) *Previous contact with cancer.* Forty-seven-point-eight per cent of the delay group had experience of cancer affecting a member of the family group, compared with 35·8 per cent of the non-delay group; 34·8 per cent of those patients who had delayed had acquaintances or "had heard of someone who had cancer and died," as against 12·9 per cent of the non-delay series. That is, patients who have had contact with cancer in the family or otherwise tend to delay (COBB *et al.*, 1954).

(iii) *Reason for initial contact with doctor.* The primary reason for seeking contact with the physician was considered to be a valuable item of investigation, as it would throw light on what each patient considered to be his critical level of endurance, above which outside help was necessary. This

* On these percentages, Confidence Limits (at the 95 per cent level) obtained from MAINLAND'S TABLES (1956), are from a lower limit of 38·8 per cent to an upper limit of 63·1 per cent; and from a lower limit of 2·1 per cent to an upper limit of 26·5 per cent respectively. This indicates there is a significant difference between the results, bearing in mind that the investigation was carried out on a particular population and not on a random sample.

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varied from case to case; from the patient who, with cancerphobia, flew to the doctor at the first sign of an ache or suspicion, to the patient with a carcinoma of the breast so advanced that she had to support the breast with her hand while she continued to scrub her floors. This item was explored, therefore, under three main headings of presentation: the lesion, a symptom of the cancer, or a completely unrelated physical disability.

Thus a patient with a cancer of the breast would be expected to present to the doctor with a lump in the breast (lesion); a patient with a cancer of the intestine or the cervix would be expected to present initially with a symptom such as pain, haemorrhage, or discharge. That this is not always so, and that patients in all three diagnostic categories can present with a completely unrelated physical disability is shown by the results.

TABLE 3.—BREAKDOWN OF CASES IN DELAY AND NON-DELAY GROUPS BY DIAGNOSIS

Diagnosis	Delay (69)		Non-delay (31)	
	Number of cases	%	Number of cases	%
Carcinoma of breast	44	63·8	19	61·3
Carcinoma of cervix	13	18·8	5	16·1
Carcinoma of colon M:	5	7·2	3	9·7
Carcinoma of colon F:	7	10·2	4	12·9
Totals	69	100·0	31	100·0

For example, as far as cancer of the breast is concerned, 95 per cent (i.e. 18 out of 19 patients) in the non-delay group, as against 59 per cent of the delay group, presented with a lump in the breast. On the other hand, 30·4 per cent* of the delay group presented to the doctor with a totally unrelated physical disorder, and only 9·7 per cent of the non-delay group. As an illustration, one patient with a cancer of the cervix, despite the symptom of vaginal haemorrhage, initially consulted her doctor because of repeated attacks of coughing and chest pain. Thus, the patient who delays apparently ignores the lesion or symptom of cancer and prefers to present on some other basis. To place oneself in the hands of a physician for some ailment far removed from the symptom or lump which might mean a fatal disease can be regarded as a compromise between the wish to seek medical help and the denial of a threat to life.

(iv) *Symptoms regarded as normal.* Only six patients disregarded symptoms of the disease, assuming that their manifestations, which they actually observed, were not abnormal. Four of these patients had carcinomas of the cervix and two carcinomas of the breast; both of them had recurring lumps in the breast at the time of menstruation over many years.

(v) *Dramatic symptoms.* As regards dramatic symptoms, there is no significant difference between the two groups. However, there are several patients in the delay series where the period of delay was shortened by a sudden haemorrhage or acute attack of pain, and there are some patients in the non-delay group where acute haemorrhage was the first symptom and produced immediate presentation to the doctor.

(vi) *Clinical delay.* In this investigation, carcinoma of the breast has been classified into three

* Confidence limits (at the 95 per cent level) of 27·5 per cent to 51 per cent and from 2·1 per cent to 26·5 per cent respectively indicate there is a significant difference between the results.
The above value indicates a significant difference between the two groups.

stages, A, B, or C, depending on the extent of the disease; carcinoma of the cervix as stages 0–4; carcinoma of the colon, including rectum, as stages A to D.

The length of temporal delay was not always related to the clinical or pathological extent of the cancer at the moment of presentation. That is to say, even in short delay periods as measured in time, there were patients with clinically advanced disease at all three sites mentioned.

On the whole, however, the longer the temporal delay, the more advanced the clinical or pathological extent of the disease. Finally, as shown in Fig. 1, there is less delay in patients suffering from a superficial cancer than an internal one.

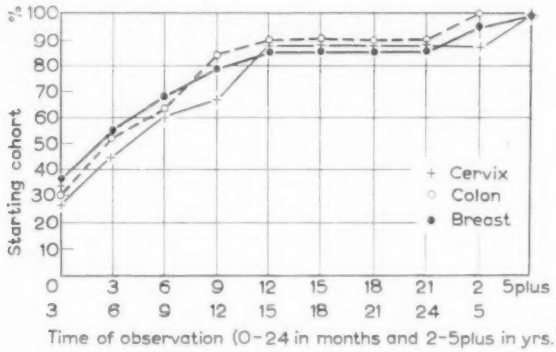

FIG. 1.—Comparison of cases of the breast, cervix, and colon by percentage of starting cohort visiting physician and time of observation.

(C) *Factors connected with the patient*

Tables 4, 5, and 5A present the breakdown of the 100 patients according to delay period and distribution by age.

The greatest number of delaying patients is found in the age group 56–65 years, and all the patients in the age group 75 years and over delayed.

It is interesting to note that there are more patients in the non-delay than in the delay group in the younger age-groups 0–45 years. Though cancer in persons under 45 years of age is relatively uncommon, patients in the younger age groups tended to present themselves early.

(i) *Ability to form a relationship with his doctor.* Sixty-one-point-three per cent* of the non-delay group, as compared with 4·3 per cent of the delay group had—and had always had—a good relationship with their doctors. Conversely, 89·8 per cent† of the delay and 19·4 per cent of the non-delay groups had been unable to form a satisfactory relationship with any physician. Subsequent case histories show that the family attitude towards medical help played an important part in the patient's decision to consult the doctor. Only 5·8 per cent‡ of patients in the delay group, in contrast to 48·4 per cent of patients in the non-delay group, had made a practice of consulting a doctor readily.

(ii) *Intelligence and education.* No difference was found between the two groups for either factor.

(iii) *Loss of an important person in the patient's life history.* It has been suggested that a state of chronic depression resulting from such a loss may predispose patients to present themselves late. However, with the exception of a few striking examples, no general evidence was obtained to support this hypothesis. On the other hand, putting aside the loss of any important person, and investigating for the presence of depressive features in the life history of the patients, distinct differences appear

* Confidence limits (at the 95 per cent level) 42·2 per cent to 77·6 per cent and 0·89 per cent to 12·0 per cent respectively.

† Confidence limits (at the 95 per cent level) 78·4 per cent to 95·1 per cent and 7·2 per cent 36·4 per cent respectively.

‡ Confidence limits (at the 95 per cent level) 1·6 per cent to 14·0 per cent and 30·1 per cent to 66·2 per cent respectively.

The above values indicate a significant difference between the two groups.

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TABLE 4.—BREAKDOWN OF 100 CASES IN TERMS OF DELAY IN CONTACTING PHYSICIAN

Delay period	Number of cases
*0–<3/12	31
3/12–<6/12	21
6/12–<9/12	14
9/12–<12/12	11
12/12–<15/12	10
15/12–<18/12	—
18/12–<2 years	1
2 years–<5 years	7
5 years+	5
Total	100

* All cases in this time period are looked on as *non-delay* for this particular study

TABLE 5.—AGE GROUP DISTRIBUTION OF NON-DELAY CASES (31)

Age (years)	Number of cases	% total cases
0–35	2	6·5
36–45	5	16·1
46–55	10	32·3
56–65	9	29·0
66–75	5	16·1
75+	—	—
Total	31	100·0

TABLE 5A.—AGE GROUP DISTRIBUTION OF DELAY CASES (69)

Age (years)	Number of cases	% total cases
0–35	4	5·8
36–45	6	8·7
46–55	19	27·5
56–65	21	30·4
66–75	12	17·4
75+	7	10·2
Total	69	100·0

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between the two groups. Two definitions of the term "depressive features" have been made: first, evidence in the life history of the patient of a state of depression which had led to either partial incapacity or to admission to mental hospital; second, evidence of a state of depression in the patient preceding, by a short period of time, the appearance of symptoms suggestive of cancer.

Forty-five per cent of the delay group gave evidence of depression as defined above, as against 29 per cent of the non-delay group. That is, patients who have had, or are having, episodes of depression tend to delay.

(iv) *Feelings of guilt.* Since it has been shown that patients who are depressed, on the whole, delay, one would expect a correlation between feelings of guilt and delay in presentation to the doctor. Our findings that a higher proportion of patients in the delay group expressed feelings of guilt about their disease is in agreement with the work of ABRAMS and FINESINGER (1953). A patient who is guilt-ridden may present himself late because he regards the illness as a well-deserved punishment.

TABLE 6.—DESCRIPTION OF PERSONALITY

Personality "type"	% non-delay series	% delay series
Emotionally labile	48·4	73·9
Rigid or compulsive	32·2	20·3

(v) *Unsatisfactory object relationships.* A history of mental conflict and broken homes appeared frequently in the family backgrounds of patients in this series. Evidence of rejection in childhood, often severe, and unsatisfactory relationships with parents were found to a greater extent in the background of patients who had delayed.

(vi) *Personality types.* To highlight personality differences in relation to delay and non-delay, a simple classification which could be applied to the majority of the patients studied was used. The patients were divided into two main descriptives groups: (1) the emotionally labile, "superficial" individual who presents his problems and manifests the origin of his conflicts at initial interview (hysterical personality); (2) the rigid or compulsive individual who hides his feelings, keeps a constant grip on himself and does not declare his conflicts easily (obsessive-compulsive personality).

Table 6 shows that the percentage of emotionally labile individuals is much higher in the delay group than in the non-delay group (73·9 per cent as against 48·4 per cent). That is, those patients who make a habitual use of repression as a psychological defence mechanism do, on the whole, delay.

(vii) *Psychiatric symptomatology and emotional maladjustment.* Fifty-six-point-five per cent* of the delay group and 19·4 per cent of the non-delay group had a history of psychiatric symptoms often long antedating the present illness. These symptoms varied from mild phobic states and depressive episodes to frank psychotic illness requiring hospitalization.

As a measure of emotional maladjustment in both delay and non-delay groups, the Culpin scale was used. This scale operates on a 4-point system as follows:

0 = No obvious psychological disorder (prior to the onset of the present illness).

1 = Minor psychological abnormalities, not sufficient to interfere with an ordinary mode of life (prior to the onset of the present illness).

2 = Psychological disorders interfering with activities but not incapacitating (prior to the onset of the present illness.).

3 = Psychological disorders temporarily or permanently incapacitating (prior to the onset of the present illness).

Application of the Culpin scale to the patients studied (Fig. 2) shows the following important points:

(1) At a Culpin "0" there is only one patient in the delay series.

(2) There is a descending incidence of psychological abnormality in the non-delay cases at the "1", "2" and "3" point levels.

(3) There is an ascending order of abnormality in the delay cases at the "1" and "2" point levels, and at point "3" the increase of delay over non-delay cases is maintained.

* Confidence limits (at the 95 per cent level) 43·7 per cent to 67·9 per cent and 7·2 per cent to 36·4 per cent respectively indicate there is a significant difference between the results.

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The overall finding, therefore, is that emotional maladjustment antedating the appearance of cancer symptoms is more common in the delay group than in the non-delay group. On graphing scales "0" and "1" together, and "2" and "3"

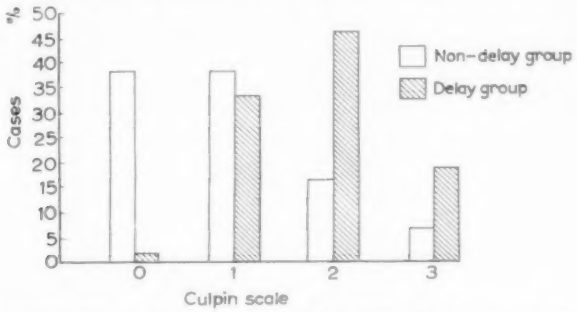

FIG. 2.—Comparison of non-delay and delay groups by percentage of cases and Culpin personality scale.

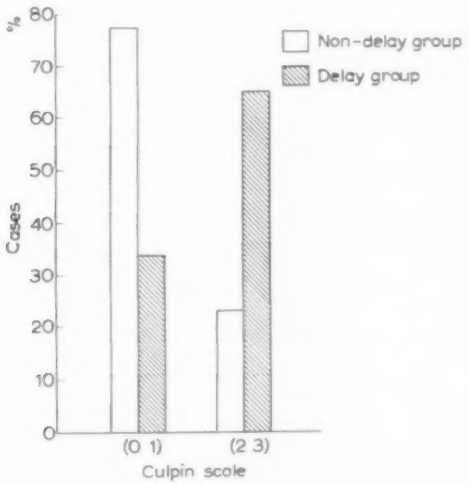

FIG. 3.—Comparison of non-delay and delay groups by percentage of cases and cutoff Culpin personality scale.

together, for both delay and non-delay groups (Fig. 3) examination of the confidence limits indicates that there is a significant difference between the two groups.

(viii) *Psychological defence mechanisms.* Cancer is a much dreaded disease; irrespective of the personality organization of the individual concerned, it is a major threat with a well founded reality basis. The presence of cancer in most people's mind is associated with expensive medical treatment, prolonged suffering, misery and mutilation at the best, and with an inevitable painful death at the worst.

Some people boldly accept the challenge and take appropriate action (the non-delay group), whereas others, rather than face up to the unpalatable truth, unconsciously or probably pre-consciously take recourse to a variety of psychological defence mechanisms.

Denial is one of the commonest defence mechanisms which may to an extreme degree defer the perception and detection of the growing cancer. Or, on the model of a phobia, rather than be afraid of the malignancy within himself (which may be incurable), the patient may project his fears into the outer world and express fear of doctors and hospitals—who and which can be avoided.

Repressive mechanisms were frequently employed by patients who delayed. These can be regarded as one end of a spectrum of psychological defence mechanisms which proceeds from the suppression of symptoms (conscious), through denial and negation (pre-conscious) to repression (unconscious). The following statement made by a patient who delayed demonstrates this continuum:

> "I put things off very often in the past—I don't think of them. I've learned my lesson, except for going to the doctor. It's all in the mind, in the subconscious mind. You lock it away in the back of your mind. You don't open it, but sometimes the door creaks open a bit, but you shut it quick."

As might be expected, in the crisis brought about by the illness, the patients were apt to use those ego defence mechanisms which they had always used.

(D) *Factors connected with cancer education*

It is of considerable importance that both the delay and the non-delay groups had been exposed and, as far as could be made out, equally, to cancer education by one or all of the methods outlined (75 per cent and 77 per cent respectively). The experience had occurred before the initial presentation to the physician and was not the result of an interest aroused by the diagnosis of the disease. Prior to the diagnosis the patients studied, with few exceptions, had been familiar with the classical initial symptoms of cancer, though some uncommon manifestations may have been unknown to them.

From this it follows that, at least in our patient population, by and large, delayed presentation was not due to "ignorance," i.e. lack of knowledge regarding symptoms suggestive of cancer. The truth is that at this time and age, everybody—rural and urban populations alike—can, if they so wish, acquire knowledge regarding the early symptoms of cancer, and that, if they do not, their ignorance is based on the unconscious belief that ignorance is bliss. As far as the actual delay is concerned, once the initial symptoms of the disease have appeared, our findings are in agreement with the observations of LESHAN and WORTHINGTON (1955), who state ". . . the great majority of patients with neoplasms have, at one time or another, had a knowledge of their diagnosis, but that this is usually repressed or suppressed." That is, we have good reason to believe that during the period preceding his presentation the patient frequently knows or at least suspects that he has a serious and potentially fatal disease but that on the principle of ignorance is immortality, as TENNESSEE WILLIAMS has put it in one of his recent plays, he keeps away from the doctor in the hope of staving off death by ignoring what may cause it.

(E) *Typical case histories*

The following case histories will demonstrate some factors connected with (i) the disease, (ii) the personality of the patient, and (iii) the influence of cancer education in leading to delay or non-delay in consulting the physician. This division is an artificial one for presentation purposes only as the three factors are obviously interrelated.

(i) *Factors connected with the disease.* This 57-year-old woman had a cancer of the rectum. The delay period was 7 months. Her early life was extremely disturbed in that she had a father who resorted to physical violence on his family, deserted them for periods up to 2 years, and on one occasion had attempted to rape the patient when she was aged 12 years.

About her illness she said, "I only thought I had piles. I thought there wasn't something very wrong. If I'd had a lot of pain it would have been different. I was embarrassed to go to a doctor. Sexual things embarrass me, and my rectum, that would be worse."

It is noted that the patient had piles from time to time throughout her life, and 19 years before she had developed a cancer of the cervix. (Note that even the previous experience of cancer in herself did not result in an early presentation.)

(ii) *Factors connected with the personality of the patient.* Case 1: Obsessive-compulsive personality. A 54-year-old woman with cancer of the breast delayed for 18 months. She was above average in

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intelligence and education. "I am very conscientious. I think it is terrible to be ill. I got the feeling that I would never be ill. I never thought it was going to happen to me. I knew that I should have gone, but there were a lot of things that I wanted to finish off. My employer, he depends on me. I didn't figure I could spare the time. We were all brought up with a sense of duty. We were not the kind of people who called a doctor for just any little thing. My father was too conscientious for his own good. He always had a project on. He had a terrible fear of hospitals. I was his favourite. I'm like him. He would never see about his ailments or anything.... I am my own worst driver. I always do my utmost. I am exact about things."

Case 2: Excessive self-love (Narcissism.) This male patient, aged 24 years, had a cancer of the rectum with secondary spread to the spine. He had delayed 10 months. The patient's attitude to the disease was: "In the first place I think why should it happen to me? Do you know what has been wrong with me? I had a cancer of the rectum. They told me it was an advanced case and they didn't know if they could save me or not. Yes, I have read about cancer. I know about persistent bleeding and things, but I've always looked upon cancer as something happening to older people, not to younger people, though, of course, I had heard of blood diseases in children. But it never seemed that this could apply to me. Sports are very important to me. I'm a very tall chap. I'm very proud of my physique. I always looked upon myself as strong as a horse. The bleeding, it didn't occur to me that it would be anything serious. Piles was a solution, so I thought of that. I thought the bleeding was a chink in the armour and shouldn't be there. I may have been frightened. I never thought it was anything serious. I couldn't picture it happening to me. I'd never been ill. I had always been strong. Nothing used to bother me. I was bigger and stronger than most of the fellows. I don't like to think that this was something wrong with me."

A man so inflated in his self-esteem, so convinced of his perfection and inviolability, could obviously not tolerate the threat which the diagnosis of a serious and potentially fatal illness constituted. In the face of it, despite better knowledge, he unconsciously warded off danger by denying its very existence.

(iii) *Factors connected with cancer education.* Case 1. Cancer education ineffective. This patient, aged 47 years, had a cancer of the cervix. Despite symptoms highly suggestive of the disease with which she was well familiar, she delayed consulting a doctor for a period of 7 months. She was a chronically anxious individual, apt to deal with threatening external stimuli by avoidance. She had postponed consulting a physician in her first pregnancy until she had reached the eighth month. She knew that she was suffering from cancer and had familiarized herself with the manifestations of the disease. Her contact with cancer educational material was extensive. She said "I have read articles in a book long before the trouble. I love watching 'Medic' on television. I just have always been interested. I always wanted to be a nurse. I think television is wonderful for telling people how things can be prevented. I think it's a way to make them know."

Case 2. Cancer education effective. The patient was a 56-year-old woman with a cancer of the breast. The time from noticing the first symptoms or signs until presentation to her doctor was 1 week. "I never had any thoughts about it. I'm not one of those whining women. I always look on the cheerful side of things. I'm easy to get along with. I'm tolerant." On discovering the lump there was an immediate increase in anxiety coped with by the erection of defence mechanisms. "I didn't sit down and brood about it. I just wanted to forget. I went out visiting more. I would read and watch television. I wouldn't watch 'Medic' after that. I was sort of preparing myself to go. I just put two and two together from what I had heard. The family doctor told me 'whenever you find a lump, see about it right away.' These 'Medics' on television tell you that when you find a lump, consult your doctor. I always read the articles in the paper. They are good."

SUMMARY AND CONCLUSIONS

The material presented shows that there is no one reason for delay on the part of patients with cancer. The largest number of delaying patients was within the age group 56–65 years, which is the period when cancer most frequently reveals itself. Patients with internal cancer delay longer than those with superficial disease, and characteristically delaying patients overlook minor symptoms.

Contrary to what might be expected, previous contact with patients suffering from cancer does not make for early contact with the physician.

Patients who delayed are characterized by poor interpersonal relationships. The importance of personal psychodynamics in leading to delay has been demonstrated. By and large, patients who delay show evidence of impaired mental health to a much greater extent than non-delaying patients. Denial, avoidance, and repression are psychological defence mechanisms commonly used by patients who delay.

Since, as could be shown, cancer education reaches both delaying and non-delaying patients to an equal extent, it is fair to assume that (a) present cancer education is as effective as would be desirable, and (b) that ignorance is not the sole factor, and perhaps not even the main factor causing delay. Cancer education must be optimistic and reassuring; to accent key symptoms of the disease in many cases increases anxiety already present in individuals. Cancer education must do more than present facts. It must reach these "isolates" in the population who are the delaying potential of the future. Such individuals are not motivated and alerted by the presents methods of cancer education.

The conclusions to be drawn from these observations and deliberations are that cancer education, by and large, succeeds in imparting knowledge regarding the symptoms of the disease but that acquisition of this knowledge in some patients fails to induce them to seek early medical assistance. The real problem, as we see it, is not whether enough information is put across to the general public, but how and how successfully this information is communicated.

As venereal disease propaganda has shown, propaganda efforts towards early presentation of patients are likely to be successful if cure of the patients on early presentation can be safely promised. This, unfortunately, cannot be done in the case of cancer. Yet there is little doubt in our minds that propaganda based on recovery is more likely to be successful than propaganda based on fear. Fear of the consequences used as a means of propaganda may defeat its objective and may foster and reinforce denial mechanisms rather than remove them.

It has been demonstrated in this investigation that much of the problem of delay is connected with the personality of the patient and his interpersonal relationships. How may the latter be improved? It is suggested that the public should receive education on the subject of cancer, not by specially organized literature or campaigns, but that the introduction should be part of the general mental health education. It should be impressed on the public that the education of parents to make their children health and doctor conscious in a positive way will make for good doctor-patient relationships in a future generation.

Finally, the physician himself. The patient-doctor contact is a situation of mutual interaction. Patients are rejected by doctors and doctors by patients because of the clash of problems, conscious and unconscious, in their respective personalities. It should be stressed, therefore, in the education of the medical student, that to know his patient and himself and improve his own interpersonal relationships is as important as diagnosing the disease. It is evident that many of the attitudes held by the public—that cancer means inevitable death, that nothing can be done—are often a reflection of the physician's feelings, who hedges over the truth by euphemistic words and phrases. These express his own feelings of failure and inadequacy in the presence of malignancy. He consequently avoids frank discussion with the patient in an endeavour to escape from the emotional difficulties that would result in them both.

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SOME PSYCHOSOMATIC ASPECTS OF CASUALTY IN REPRODUCTION

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NOTHING in biology is due to chance. However, a large range of phenomena connected with the reproduction of species, and notably of our own, remain unexplained. Implicitly they are attributed to chance, or—the same concept once removed—to a marginal inefficiency of biological processes accepted as inevitable. One may list the chief of these unexplained phenomena: termination of healthy pregnancy by miscarriage, stillbirth or neonatal death; congenital malformations; weakly, disease-prone infancy suggestive of "weak constitution"; failure of normal mental development; behaviour disorder, hypersensitivity or instability of temperament which would also seem to be "constitutional."

If these phenomena were extremely rare yet were observed to recur in consanguineous unions, they could be accounted for by the concatenation of recessive genes in both parents (the term "gene" being defined here, to save controversy, as the hereditary element, whatever that may be, in the determination of a characteristic). The rarer a defective recessive gene, the less likely is it to occur in both partners of any one mating; the longer therefore can it be carried without manifesting, and destroying, itself. The incidence of phenylketonuria, a type of mental defect due to a recessive gene, has been estimated at 1 in about 50,000 in the United Kingdom, and 1 in 25,000 in the United States (PENROSE, 1949). By contrast, the incidence of cleft palate has been found to be between 1 in 600 and 1 in 1000 (HARRIS and ROSS, 1956); congenital heart disease has been found to occur naturally in rats and several other mammals at the rate of about 2 per cent (SOBIN, 1954).

Congenital malformations, although showing a certain hereditary tendency in some cases, fail to conform in their distribution to either a dominant or a recessive pattern (WARKANY, 1947; PENROSE 1951). A way out of this etiological dilemma was to identify the same malformation as due sometimes to a dominant, sometimes to a recessive gene and sometimes—when hereditary antecedents were completely lacking—as a fortuitous "phenocopy." This hypothesis of the phenocopy has low methodological status as an unverified explanation of exceptions to the favoured general theory.

The theory of penetrance is another special explanation employed to patch up the inadequacy of traditional genetic theory: if the incidence of a malformation presumed to be due to a dominant gene is too low to fit the standard dominant pattern, this is explained by hypothesizing that the influence of gene "penetrates" in only a proportion of the individuals. But why does the effect of the gene fail? "Penetrance" adds nothing to our understanding of the process; it merely restates the lack of law by a metaphor the meaning of which for gene-action is intangible and unverifiable, and we are thrown back to the pseudo-hypothesis of indeterminacy.

The experimental production of malformations in animals has helped to elucidate this riddle of occasional manifestation. FRASER and FAINSTAT (1951, 1954) produced cleft palate in mice at frequencies of between 4 per cent and 100 per cent, according to the genotype of the mother and of the foetus. Feeding rats on a vitamin A deficient diet, ANDERSON (1941) observed that in one strain, in which diaphragmatic hernia occurred spontaneously in 2·7 per cent of the offspring, the incidence of the malformation rose to 19 per cent; in another strain, in which this malformation was normally completely absent, only 0·9 per cent were affected. These experiments and much other similar work point to a hypothesis of a genetic proneness facilitated by exogenous stress (ANDERSON, 1941). This would explain both the observed hereditary tendency in malformation and also the lack of Mendelian pattern, and makes the special explanations such as those of the phenocopy or of penetrance superfluous.

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The hypothesis of facilitation opens up two lines of enquiry: the first is the nature of the stresses which can produce malformation, mental defect and proneness to infantile ailment in human beings; the second is the biological role of comparatively frequent facilitated defects which should, by the laws of survival, have been eliminated.

It cannot of course be assumed that the same insults produce malformations in human beings as in animals. However, the findings of the experimental work upon non-human vertebrates have a certain consistency to which it is reasonable to suppose human beings are no exception. The first is that effective stresses vary surprisingly even between closely related mammals; thus cortisone produces malformations in mice but has no effect, however great the dosage, in rats (SOBIN, 1954). It is therefore possible that human beings respond by malformation to stresses to which animals are immune or which have not been the subject of experiment; this means that we must be prepared for surprises.

A further feature of the work upon animals was that if the stress applied was too severe the result was not malformation but resorption of the foetus or still-birth (SOBIN, 1954; WARKANY, 1947; FRASER *et al.*, 1954). Malformation would therefore appear to be at the borderline of stress-tolerance. WARKANY (1947) points out that general starvation before conception leads to sterility; in early pregnancy it results in embryonic death or in miscarriage. Revolving a pregnant rat in a drum, SOBIN (1954) found that congenital heart disease was produced in the offspring at 200 revolutions, but that at 800 revolutions the incidence of the malformation could not be ascertained owing to a large degree of resorption.

The concept of malformation as the result of borderline stress is confirmed in human beings by the work of LILIENFIELD, PASAMANICK and their colleagues (1951, 1954, 1955, 1956a, b, c) in relating mental deficiency, cerebral palsy, epilepsy and behaviour disorder to complications of pregnancy and delivery. The same factors were found to be even more significant for infant loss. This led them to the hypothesis of a "continuum of reproductive casualty," in which the surviving mentally or physically handicapped child is at the just sub-lethal borderline.

Insofar, in short, as the evidence is at hand, the general biological picture would seem to be the same for human and non-human vertebrates, in the sense, first, that malformations are the result of genetic proneness facilitated by a variety of stresses, some of which are specific to each species, and second that they stand at the borderline between reproductive failure and success.

The reporting by GREGG (1941) that German measles during pregnancy could result in mental deficiency and/or congenital malformation stimulated a search for other and more frequent causes. The studies of LILIENFELD, PASAMANICK and their colleagues had to rely upon obstetric records of some years back, which only recorded complications known at the time of the birth. The few complications of pregnancy which were included, notably toxaemia, bleeding and those of delivery which presupposed pregnancy-complication (e.g. breech and other malpresentations) tended to be significant, while other forms of difficult birth were not. But toxaemia was a prelude to only 3 per cent of the cases of cerebral palsy and 8·2 per cent of the mental defect as opposed to 6·9 per cent of the normal controls. In the sample of 102 mentally retarded children (STOTT, 1957) referred to below 21·5 per cent of the mothers had miscellaneous ill-health during pregnancy compared with 11·3 per cent among the controls. None of these was German measles, and only one—a severe cold—could conceivably have been a virus infection. In a further sample of 365 mongol and 484 non-mongol defectives only 6 cases of German measles, 10 of influenza and 9 of other infectious diseases were reported. There is elsewhere some evidence that mumps and influenza can produce malformation, but this is so far

inconclusive. It must be admitted that attempts to relate mental defect and malformation to illnesses during the pregnancy have been disappointing. We have consequently been left in the tantalizing position of suspecting the existence of noxious factors operating during the pregnancy without being able to track them down.

An increase in the frequency of congenital malformation, together with significant falls in birth weights, was reported from many parts of Germany during the immediate post-war years (NOWAK, 1950; ARESIN and SOMMER, 1950; GESENIUS, 1951–2; KLOTZ, 1952; EICHMANN and GESENIUS, 1952; WORM, 1952; GREBE, 1953). Analysing the figures from the Municipal Clinic at Chemnitz, where the incidence of malformation in the years 1946–9 was ten times that of the economic "good years" 1934–8 and 2½ times that of the "bad years" 1931–3, NOWAK (1950) shows that this cannot be explained by the possibly greater age of the post-war mothers. There would seem to have been too few mothers in the older age-group for valid comparison, but the increases are equally apparent in the 22–35 year-group. NOWAK, together with EICHMANN and GESENIUS in Berlin (1952) and ARESIN and SOMMER in Leipzig (1950) note that the increases were most striking among malformations of the central nervous system, and indeed these seemed to account for practically the whole of the gross increase. The Berlin study showed even more strikingly to what extent the post-war increases were limited to the neuro-ectodermal malformations (anencephaly, spina bifida, hydrocephalus, meningocele). This group averaged 6·5 per 1000 births during the years 1946–50, reaching a peak in 1949 of 7·6 (104,076 births) compared with 2·58 per 1000 during the six war years 1940–5 (139,418 births). Between 1925 and 1932 the rate had been remarkably constant so far as can be judged by the smaller number of births covered (44,351)—at between 1 and 1·4 per 1000—but went up to 2·2 in 1933, the year of Hitler's coming into power. Between 1934 and 1939, after a slight drop, it rose steadily, averaging 2·29.

This type of malformation seems consequently to act as a barometer of social security, or probably more accurately, of the incidence of intense hardship and anxiety within a community or a section thereof. As part of the above study 100 mothers of malformed children and 100 controls were interviewed soon after the births to obtain histories of the pregnancies. Forty of the cases reported amenorrhea during several months prior to conception, as against 32 of the controls; but of the 40, 28 had infants with neuro-ectordermal malformations. When asked to what they attributed the malformations, 50 of the mothers blamed *Seelische Aufregungen* (emotional upsets), 29 more gave this cause together with continuous food shortage, and only 15 blamed the latter alone. Unfortunately, corresponding figures for the controls are not given. German authorities are agreed in attributing the post-war crop of malformations to general malnutrition in conjunction with psychological (*seelisch*) stress, although no systematic studies are to hand. In the tabulation of malformations reported in the literature which were attributed to emotional stress with or without malnutrition) GREBE (1953) includes mongolism, dysplasmatic mental deficiency, anencephaly, cleft lip and palate, malformed limbs and other skeletal anomalies.

Little attention was paid to the incidence of malformation during the war itself, probably because the data were not recorded so systematically, but ARESIN and SOMMER (1950) give the figures for Leipzig for the whole period 1936–48. Their object was to show the increase during the early post-war years, but in fact this started from the beginning of the war. During the years 1936–9 inclusive the average incidence

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was 0·72 per cent (109 cases) and for 1940–5 inclusive 1·07 per cent (241 cases). This gives a $\chi^2 = 13{\cdot}7$ and less than a 1,000 to 1 risk of chance. The importance of this finding for the present discussion is that there would have been little or no increase in malnutrition during the war itself, and indeed the incidence jumps from 0·76 per cent in 1939 to 1·17 per cent in 1940. This would seem to point to the general increase in the risk of shock, anxiety and other socio-psychological events as the etiological factors.

The acceptance in Germany of psychological stress as a factor in reproductive capacity arises from anatomical studies both upon animals, and from post mortems by STIEVE of women imprisoned and subsequently executed in recent years, presumably during the Hitler regime. GESENIUS (1951–2), writing in 1951, summarizes the current view thus: "most workers are to-day inclined to attribute to the nervous system an influence upon the developing embryo". . . . (While old wives' tales must be discounted). "It must be regarded as proved that injury of the male, and still more of the female organs of generation is possible from nervous shock. Medical practice after the first and still more after the second world war has had experience of amenorrhea in connection with the year's land-service, work conscription, imprisonment, flight, hardship and hunger. Stieve in particular has provided in recent years the anatomical confirmation for neural injury to the human ovary, while he demonstrated some decades back in extensive experiments upon animals the influence of the nervous system upon the reproductive organs. Certain difficulties are to be sure raised by the question whether such neurally damaged ovaries remain fertilizable, as a great part of the follicle is usually seriously affected. We have therefore, with Bondi, to think in terms of borderline cases of injury, that is, of still fertilizable eggs which have suffered moderate damage."

It is interesting to note once more, in this suggestion of a psychosomatic etiology, the recurrence of the concept of borderline insult. It would mean that in verifying this, as any other possible cause of malformation, we must expect to find the insult resulting on the one hand in sterility or miscarriage, and on the other in completely normal offspring, with possibly only a small intervening group of affected but surviving children.

KLOTZ (1952) reported two cases in which there was no question of inadequacy of diet, and in which there was a clear heredity and good health during the pregnancy. One of these concerned a 22 year old woman who early in her second pregnancy was forced to flee for political reasons from her own country, and gave birth to a mentally defective girl. The second links up more closely with the work of STIEVE in that the psychological trauma—the news of the husband's being wounded—occurred some three weeks before conception. The woman received a second shock at the beginning of the fifth month on hearing her husband had been wounded a second time, but Klotz rules this out as being past the organogenetic stage of gestation. The woman gave birth to an anencephalic. Subsequently she had two normal children. In justification of his attribution of the malformation to the shock prior to fertilization he quotes STIEVE: "If at the moment at which the insult (sudden distress, fright) supervenes, there is in the ovary a follicle ready to emerge or almost so, it does not burst but the ovum collapses and the whole follicle degenerates." (STIEVE 1942).

Psychosomatic stress during the pregnancy itself as a possible etiological factor in reproductive casualty has been the subject of little systematic investigation up to the present. There are four probable reasons for this omission. The first is that the great advances in genetics during the past century have placed a premium upon explanations in terms of heredity, and it has been only very recently—since GREGG's

rubella discovery in 1941—that attention has been paid to environmental influences in general. The second is that, in face of the medical stand against old wives' tales, the worker who gave credence to shocks, mental states and suchlike folksy data laid himself open to the taunt of being unscientific. The third is that workers are inclined to sheer off variables, such as mental states and shock, which are hard to measure and are difficult to define and to quantify. But, as TINBERGEN (1951) points out, all new discovery has to employ relatively inefficient techniques, since those best adapted to the handling of the new sorts of data have yet to be perfected. Lastly, psychosomatic influences are rejected because it is difficult to see how they could operate upon an apparently well-protected foetus.

Nevertheless a certain amount of evidence is accumulating which suggests that systematic study of psychosomatic influences may be worthwhile. INGALLS (1947) reports a case of a healthy woman of 36 who gave birth to mongoloid twins. At about the sixtieth day of pregnancy she was involved in a collision with another car on a mountain road and suffered moderate shock. Within a few hours she had a slight haemorrhage, and stayed in bed for nearly a week on her doctor's advice.

LEVY-SOLAL and his colleagues (1952) made a study of 69 pregnancies resulting in major malformations by interrogation of the mother after the birth; they took 100 control-pregnancies which they observed concurrently. There were slightly more infections and much more toxaemia among the controls; the only factor notably more frequent among the malformed were *frayeurs et émotions subites* (frights and sudden emotions—i.e. the categories I and II in Table 1 below), of which there were 8 compared with 3 among the controls. If an "expected" of 4·5 instead of the minimum 5 in one of the cells is allowed, this gives a χ^2 of 4·72 ($p < 0{\cdot}05$). Apparently no record was made of continuous anxiety or distress.

In studies carried out recently in Bristol and from questionnaires returned by members of the National Society for Mentally Handicapped Children, a much greater incidence of psychosomatic factors was reported in respect of the defective children than among their siblings and other controls (Table 1). In order to diminish the inevitably subjective nature of such influences, they were rated by objective *situational* criteria. That is to say, a situation or event was rated as distress- or shock-producing if this would have been its expected effect upon an emotionally stable woman. This naturally under-stated the degree of actual anxiety and worry, as a certain number of the women, who admitted in the interviews or questionnaires that they were "worrying types" would have become upset and harassed by everyday difficulties.

The purpose of this study was to take soundings as to a number of possibilities rather than rigorously to test the reality of certain causes, and so the data must be regarded in this sense. Since the questionnaires were mostly anonymous it was not generally possible to obtain further details when the parents merely answered questions about shock or other stress in the affirmative without stating their nature. In order that this element of uncertainty should not detract from the more specific reporting of the stresses, the unspecified stresses are given separately (columns Ia and Va) and all the data are treated both counting and discounting these. It will however be noted that the unspecified stresses follow the same type of distribution as the specified ones, and indeed in so far as they represent attempts to induce abortion may be more real. The large category of unspecified stress (Va), with its heavy preponderance among the defectives, suggests that it represented either an anxiety-state of the mother or unadmitted distress at the discovery of pregnancy with possible attempts at termination. No question was asked in these questionnaires as to whether the mother became upset at knowing she was pregnant.

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TABLE 1.—INCIDENCE OF PSYCHOSOMATIC STRESS IN PREGNANCY

	(a) Mongol	(b) Non-mongol	Totals for defectives	(c) Siblings of (a) and (b)	(d) Children of above average intelligence	(e) Children of professional caseworkers	Totals for normal controls
Total at risk	365	484	849	87	111	146	344
(I) Shock from accident, severe fall, bomb-shock, fright, attack, calamity to third party, bad news of member of family of whom mother was fond	55	58	113 (19·8%)	2	2	18	22 (6·4%)
(Ia) Unspecified shock	25	22	47	—	—	—	
(II) Anxiety over serious illness of or grief at death of husband, child, wife's parent, sibling (not counted in I)	27	34	61 (7·2%)	5	4	8	17 (4·9%)
(III) Marital trouble (infidelity of husband, etc.)	3	8	11 (1·3%)	—	—	—	0 (0·0%)
(IV) Personal difficulties in connection with sharing accommodation; disagreements with in-laws; threat of eviction; fear of loneliness (ordinary housing difficulties excluded)	11	17	28 (3·3%)	2	3	1	6 (1·7%)
(V) Miscellaneous specific anxieties (husband's loss of job, business difficulties, etc.) distress at becoming pregnant; difficulty with other defective or maladjusted child)	16	13	29 (3·4%)	4	3	5	12 (3·5%)
(Va) Unspecified harassment or distress	19	21	40 (4·7%)	1	1	2	4 (1·2%)
(Vb) General housing difficulties	19	39	58 (6·8%)	9	6	16	31 (9·0%)
(Vc) Husband serving in war	10	23	33 (3·9%)	3	4	14	21 (6·1%)
Totals for specified stress (I—V)	112	130	242	13	12	32	
Totals for stresses (I—V), Ia and Va	156	173	329	14	13	34	

Shock was defined as some sudden cause of mental stress, which might also be accompanied by sudden physical stress as from an accident. It was often difficult to say whether the stresses of category II fell within this definition, because it might not be always made clear whether the news, for example, of the death of a loved relative was sudden or anticipated. The same would apply to the infidelity of the husband. The items of the first three categories, all of which would entail at least some degree of acute mental stress whether sudden or not, are seen to be preponderant among the pregnancies antecedent to mental defect. The same applies to those of category IV, and here again there may have been times when the mental stress owing to personal upsets was acute. On the other hand, the miscellaneous, less acute anxieties of category V show a more or less equal incidence in relation to the defectives and normals; the difficulties experienced with the earlier defective child—which would be confined to the normal controls—were not numerically large enough to give a false weighting.

Categories Vb (general housing difficulties) and Vc (the absence of the husband on war service) were included by way of a "control" of normal anxiety such as would be shared by large numbers of expectant mothers during and after the war. Both were more prevalent among the normal controls than among the defectives. One obvious reason for this is that housing difficulties which caused distress in the form of bad human relationships, and the husband's being actually wounded, reported killed or missing, etc., would have already been subtracted from these totals in respect of the defective groups as they would be in categories II and IV. It seems safe to assume that there is no evidence at all for such widespread general anxieties having a harmful effect upon the foetus.

To give an idea of the nature and degree of the shocks included in column I of Table 1, the 55 for the mongol group are itemized as follows:

TABLE 2

Involved in street accident	1
Serious falls	12
Other accidents	4
Death of own child	2
Death of one of mother's parents (3) death of brother (1)	4
Sudden illness, accident to husband, sister (suicide)	2
Other bad news, etc. (feared and actual) accident to daughter, told pregnancy must be terminated, heard boy of close friend had incurable cancer	4
Shocks—electric (1) gas explosion (1) fire-incident in house (3) firework in cinema (1) burglars (1) attacks by dogs (5) from dead rat (1) witnessed suicide attempt (1) roof collapsed while asleep below (1)	15
Bombed out, shocked in bombing incident	11
TOTAL	55

The inclusion within the above table of any particular shock does not mean that its etiological significance is assumed: once the situational criterion given above was adopted, all incidents, at whatever period of the pregnancy, had to be included. Nevertheless, on purely actuarial grounds, the frequency of such incidents within a limited number of phases of some 9 months suggests etiological significance for a number of them.

Whatever stresses in the form of shock may facilitate mongolism, they must occur—barring for the present the possibility of degenerative change in the foetus—within the first three months of gestation. This would not be generally known to the mothers, so that if they reported the stresses as occurring in the early period this would serve further to confirm the etiological reality of the events in question. The histogram in Table 3 gives the stage of the pregnancy of the shocks as included in Table 1, columns I and Ia, in respect of mongols and non-mongol defectives. More than one incident or form of stress was reported for certain pregnancies and so the shocks are given, first, when they are the sole stress reported and, second, when they occur along with other mental stresses (those of columns I–V in Table 1). Where the mother reported shock "at 3 months," this was counted as during the fourth, and so on; for this reason, and also to allow for inexactitude of memory or not knowing the precise stage of gestation at the time, the first 4 months are treated as the critical early stage.

It cannot of course be presumed that the shocks in question were responsible for all or any of the mental defect; a proportion of them at any rate must be reckoned to be fortuitous. Neither can it be supposed that the mothers would generally have recalled accurately to within a month. For example the 6th month seems a strong non-favourite, probably because of the popular emphasis

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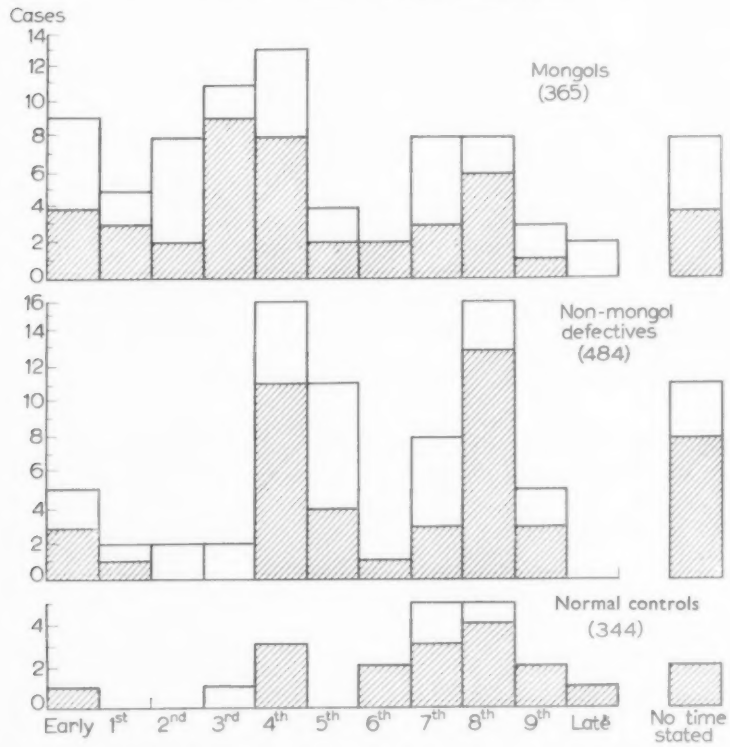
upon the 7th ("At 7 months" being put in the 8th), which is also shown among normal controls. The peaks at the 7–8th months both for the defectives and normals may be due to the mothers in general associating traumatic events more vividly with their condition at later than at the early stages. Nevertheless the comparative incidence is suggestive. There are distinct peaks in both defective groups for the 4th month, which would, as explained, include all those who stated "at 3 months," and it is possible that this has some significance even for the non-mongols. The most striking difference between these two groups however is the consistently greater prevalence of shocks

TABLE 3.—STAGE OF PREGNANCY SHOCK



Shaded portions show the proportion of shocks which were the sole factor in any one pregnancy

in the "Early" and first 3–4 months among the mongols, which is even more marked among the "sole factor" shocks. This is consistent with the general attribution of mongolism to insult during the first 3 months; whereas some of the non-mongol defect would have been due to birth injury and anoxia and to post-natal causes such as meningitic infections, early pneumonia, whooping cough and burns or other accidents. It is also possible that traumatic events during the later stages of pregnancy may damage the brain cells of the foetus or retard development other than by what is regarded as malformation.

In assessing the significance of this preponderance of shock during the early period among the mongols there was a small statistical complication in that some of the mothers omitted to give the stage of pregnancy, and the numbers were insufficient for a 6-cell arrangement. But it is permissible to split up the "No time stated" group equally between the early (first 4 months) and the late (5th to 9th) periods on the assumption of a chance distribution as this would weight the result somewhat

against the hypothesis of significance. This is done in the following table, with the indefinite group apportioned as shown in brackets.

TABLE 4.—STAGE OF PREGNANCY OF SHOCK

(a) All shocks				(b) "Sole factor" shocks			
	"Early" + first 4 months	"Late" + 5–9th months			"Early" + first 4 months	"Late" + 5–9th months	
Mongols	46 + (4)	27 + (4)	81	Mongols	26 + (2½)	14 + (2½)	45
Non-mongol defectives	27 + (5½)	41 + (5½)	79	Non-mongol defectives	15 + (4)	24 + (4)	47
	82½	77½	160		47½	44½	92
$\chi^2 = 6{\cdot}81$ $n = 1$ $p < 0{\cdot}01$				$\chi^2 = 4{\cdot}70$ $n = 1$ $p < 0{\cdot}05$			

It is seen that the tendency for more shocks to be reported in the early stages of pregnancies antecedent to mongolism compared with non-mongol defect is significant at the 100 to 1 risk of chance when all shocks are considered, and 20 to 1 risk with the shocks as sole reported factor. The slightly lower significance among the sole-factors is due entirely to smaller numbers, the proportion of the "Earlies" among these being almost exactly the same. The most that can be inferred from these figures is that, together with other factors such as genetic proneness and the general state of health, neurophysical robustness and age of the mother, the above traumatic events may possibly act as precipitating factors both in mongolism and other so far unclassified defect. The corroboration of this possibility must await more systematic study. The number of cases is also insufficient to show which of the various types of stress are most likely to be injurious. In subsequent studies a broad distinction might be made between shocks which were primarily physical, and those which involved mental stress. In the present study ordinary falls were ignored, but bad falls and other accidents were included in the "Shocks" of Table 1, column I. Among the mongols these did not predominate in the early period as did the remaining, that is to say, the psychosomatic shocks, but did so more than among the non-mongol defectives and the normal controls. There is evidently a strong tendency for falls either to occur or to be reported during the later stages. The distribution is shown in Table 5 below.

TABLE 5.—STAGE OF PREGNANCY OF PHYSICAL SHOCK
(SEVERE FALLS AND ACCIDENTS)

	First 4 months	5th–9th months	Totals
Mongols (365)	7	9	16
Non-mongol defectives (484)	2	10	12
Normal controls (344)	1	9	10

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Whereas this would suggest that physical shocks are of less significance than mental shocks, there would seem to be a possibility that some of the former might be associated with mongolism. It is after all difficult to draw a firm line between the physical injury, say in a street accident, and the element of fright or fear. The above figures are in addition of some interest for the assessment of the reliability of the mothers' reporting: the incidence of physical shock is almost exactly the same among the normals and the non-mongol defectives, and only slightly greater among the mongols, which suggests that if there is any retrospective distortion of memory it does not operate on a large enough scale to account for the preponderance of psychosomatic stresses among the defectives shown in Table 1. Among happenings during the pregnancy mothers tend to think first of "nasty falls," so that this category of shock might have been expected to show the greatest unreliability on account of memory as between the defectives and normals.

The above tables reported the incidence of traumatic events irrespective of the number of pregnancies involved. Tables 6 and 7 below compare *the number of pregnancies* subject to some kind of psychosomatic stress (as per Table 1) for each

TABLE 6—NUMBER OF PREGNANCIES INVOLVED

	All stresses		Specified stresses only (excluding Ia and Va)	
(a) Mongoloid defectives (365)	127	34·5%	90	24·6%
(b) Non-mongol defectives (484)	143	29·5%	108	22·3%
(c, d, e) All normal controls (344)	57	16·7%	53	15·4%

TABLE 7.—COMPARISONS BETWEEN GROUPS

	All stresses		Specified stresses	
	χ^2	p	χ^2	p
Mongoloid defectives/normal controls	31·0	<0·001	9·4	<0·01
Non-mongol defectives/normal controls	18·3	<0·001	6·21	<0·02

group of defectives and the normal controls. The comparisons are made both in respect of all reported stresses (i.e. including columns Ia and Va in Table 1) and for specified stresses only (columns I–V).

It is seen that there is a significantly greater incidence of psychosomatic pregnancy-stresses among both groups of defectives compared with the normal controls.

If the stresses in question play some part in the causation of the mental defect, this would be an expected result. On the other hand it can be assumed that part of this preponderance is due to the mothers of the defectives having remembered incidents better, so that these figures in themselves serve once again only to establish the need for more systematic enquiry. Nevertheless the specific and unusual nature of many of the shocks and other causes of distress makes it hard to dismiss them as events which would be likely to occur in a random sample of pregnancies but which the mothers of normal children would forget. It is moreover seen that there is a somewhat higher incidence of stress among the mongols compared with the non-mongol defectives; if, as assumed above, some of the latter would be due to post-natal causes, this would again be an expected result if the stress-hypothesis has some basis in reality. There is after all no reason to believe that the mothers of mongol children would be less likely to recall stresses than those of non-mongol defectives.

There is further indirect evidence pointing to the reality of the psychomatic stresses arising out of the association previously reported (STOTT, 1957) between pregnancy stress and, on the one hand, malformation and, on the other, non-epidemic illness in the child under the age of 3 years. These associations were found consistently in a group of 102 subnormal and defective children, and also among five control-groups totalling 450 children. That between pregnancy stress and early ill-health was also significant at less than 100 to 1 risk of chance ($\chi^2 = 7{\cdot}33$) among the 484 non-mongol defectives of the present study, even though the information as to non-infectious illness in the mother during pregnancy was scanty. It was noteworthy, however, that with precisely comparable information for the mongol defectives this association was completely absent among them, χ^2 being nearly 0. This could be taken to mean either that, for mongols, the totality of stresses had no etiological significance as far as the early illnesses of mongols were concerned, or that they had significance not for the illness alone, but for the whole syndrome of mongoloid defect, of which proneness to illness is a part. The hypothesis of no etiological significance is contradicted by the findings of earlier workers, notably of INGALLS, who found a significant proportion of bleeding in the early months; in the present sample many mothers reported "threatened miscarriage," sometimes immediately following the shock, and this was counted as a stress. It would also seem improbable that a heterogeneous group of stresses should be etiologically connected with reproductive risk, and notably with early ill-health and malformation, and yet not be connected with mongolism (which must be regarded as a type of malformation). On the other hand, if environmental stresses played some part in mongolism as a whole (rather than in those who had serious ill-health under 3 years) no association within the mongol group between stress and ill-health would be expected.

The sequel of juvenile ill-health serves to fill out the related hypothesis of borderline insult (WARKANY) and of the continuum of reproductive casualty (LILIENFELD, PASAMANICK *et al.*). It represents a very wide band at the extreme edge of the spectrum of casualty. Thus, while in the studies reported earlier (STOTT 1957) there were only 22 malformations in the combined retarded samples of 552 children (18 of them associated with reported pregnancy-stress), there were 137 cases of early illness (83, or 61 per cent, after known pregnancy-stress).

The gist of these findings is that the foetus would seem to be far more vulnerable to outside influence than had hitherto been supposed.

This not only runs counter to the traditional view, but also requires biological explanation. By the crude theory of the survival of the fittest, proneness to malformation and to ill-health should have been eliminated during the rough-and-tumble of the millenia of savagery. The above psychological trauma would notably have been of much more frequent occurrence among primitive man than in our present-day civilisation, despite the hardships and hazards of war; and it goes without saying that the presence of a malformation or of congenital weakness of health would be detrimental to the survival of the individual concerned. Students of the behaviour of the social animals, among which man is included, have however noted responses which are undoubtedly detrimental to the individual's survival but which contribute to the survival of the group. All that behaviour termed "courageous" belongs to this class. We may therefore ask whether the persistence of malformation and juvenile ill-health may be explained by their having survival value for the *group*. Just as the occasional male may have to be sacrificed in battle in defence of the herd, may there not be circumstances which demand sterility, miscarriage or the sacrifice of the infant? To put the question more succinctly, is the "continuum of reproductive wastage" phylogenetically a mechanism of fertility-regulation? That some mechanism must exist among food-gathering peoples has often been borne in upon anthropologists. To assume that the population is regulated by sheer starvation does not fit the facts, for there is little evidence of chronic undernourishment in such communities, the individual savage being a well-developed specimen capable of leading a strenuous life. If this argument is valid, there would be great survival-value to the group in some form of fertility-regulation which by-and-large adapted the size of the herd or tribe to the means of existence without reducing the functioning efficiency of its members by malnutrition. This might be achieved if the reproductive rate itself were in some way responsive to the contemporary food situation. Actual malnutrition during the pregnancy would

thus be expected to lead to sterility and infant-loss, but such could only occur when the dangerous state of a general semi-starvation was already upon the tribe. Consequently some much finer barometer of shortage would be needed than the fact of starvation itself. Increasing difficulty in finding food would entail foraging further afield, the hardship of treks, clashes with other tribes involving death and injury of some of the menfolk, retaliatory raids during which children might be killed or maimed, unforeseen natural dangers, the terror of strange places, possibly quarrelling and a straining of personal relationships within the tribe. To the risk of general food-shortage must be added the particular difficulties of a family deprived of the adult male hunter by illness, injury or death, which would make an additional child a burden. These calamities constitute just those types of harassment, distress and shock which were noted in the above study. On biological grounds, therefore, it is feasible that psychological stresses of this character could be an agent limiting fertility.

This supposition is borne out by two further observations which have previously been accepted as natural phenomena requiring no specific explanation. If, as would appear, the greater part of non-epidemic ill-health in the early years is "facilitated" by pregnancy-stress, the higher disease- and death-rate among male infants lends itself to the explanation that the recovery-rate of a tribe would be less prejudiced if it was mainly the male infants who were sacrificed in times of temporary hardship. There is also a greater incidence of other types of impairment among males. The second piece of corroboratory evidence is the surprising tendency of weakly infants, with devoted care, to "grow out" their ailments and to become sturdy adolescents—hence no doubt the folk belief that a child's constitution changes at the seventh and fourteenth years. When it is considered that juvenile ill-health is at the very edge of the continuum of reproductive casualty, and presumably reflects the milder pregnancy-stresses, we can see this constitutional resilience as a means of countermanding the "death-sentence" upon an infant born in hard times, provided conditions improve to the extent of allowing the mother to nurse the child through the delicate early phase. If on the other hand the mother is so preoccupied or exhausted as to be forced to deprive it of the special care needed, then it is likely to become a "casualty" or to develop personality-characteristics which have the effect of enstranging it from the group. It may be that BOWLBY's thesis as to the effects of maternal deprivation will receive clearer confirmation when linked with heightened mother-need due to congenital ill-health and/or "sensitisation" of temperament consequent upon pregnancy-stress.

The above theory of fertility-regulation would apply in a general way to mental impairment following unfavourable pregnancy conditions in that mental handicap and slowness of reaction would increase the risk of misadventure in a primitive community. All such linkage of the present tentative findings to general principles of ecological interaction must at this stage remain speculative. There remains however the intriguing query as to the extent to which mental development even within the normal range is conditioned by foetal development. It is probably true of all animals, who feed upon other living matter—as opposed to the plants who obtain their food chiefly by chemical conversion of inorganic materials—that any species which becomes too efficient destroys its own food supply or if a microbe its host, and so destroys itself. If primitive man, as an animal with a highly developed brain as his speciality, became too efficient, this would apply to him also. Any process which tended to lower the mental capacity of a significant number of individuals in times of food shortage would therefore achieve the paradoxical effect of improving the group's chances of survival.

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SUMMARY

Reproductive casualty has for long been regarded as a matter of heredity, chance or of an inevitable marginal inefficiency of biological processes. Despite recent findings as to the effects of physical complications of pregnancy, the greater part of the casualties remain unexplained either upon a hereditary or an environmental hypothesis.

Experiment with animals has revealed an interesting new relationship between these two factors in that hereditary proneness is shown as facilitated by environmental stress. Congenital malformation and other handicap of surviving offspring appear at the sublethal borderline of a continuum of reproductive casualty.

Very little attention has been given hitherto to the effects of psychosomatic stress, this being a topic which has fallen under the taboo of " old wives' tales." The general increase in malformations in post-war Germany stimulated wider enquiry into causes; cases were quoted from the German literature of shock producing reproductive casualty. In America a case of mongoloid twins was attributed to a car accident.

In a survey by interview and questionnaire, shock and other psychosomatic stress in the form of distress and harassment of the mother, etc. were found to be significantly more frequent in the pregnancies antecedent to both mongoloid and non-mongoloid defect. A number of checks were made of the consistency and reliability of the data, namely the higher incidence of stress reported by the mothers of mongol compared with non-mongol defectives, the preponderance of stress in the early stages in the mongol group, the association between pregnancy-stress and early ill-health except, significantly, among the mongols.

These data do not in themselves establish beyond doubt the etiological reality of psychosomatic influences during the pregnancy, but indications from several sources would seem to point to the need for wider and more systematic studies of such influences, even though such research offers considerable methodological difficulties.

Finally, a reason was sought for the biological persistence of reproductive casualty. It was suggested that this acted as a fertility-regulator. Serious malnutrition would no doubt result, as in animals, in sterility or in the loss of the offspring, but the stresses resulting from unsettled conditions arising from mere scarcity might give rise to borderline handicap which would reduce fertility less drastically and allow for recovery if times improved. In this way casualty resulting from psychosomatic stress might have survival-value for the group.

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GUIDE TO PSYCHOSOMATIC LITERATURE IN GERMANY SINCE 1945

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THE aim of the following survey is to make it easier for those interested to find their way amongst the German literature on the subject of psychosomatic medicine. It makes no attempt at completeness, but endeavours to gather together the most important items. We are aware that this implies a subjective standard. Moreover, amongst the great number of publications, it may well have happened that one or another has escaped us, which ought in fact to have been mentioned.

Since the wealth of the material prohibits the making even of brief reports on individual works, the aim of any guide to our psychosomatic literature can only be achieved by means of a classification of the material. This question has caused us much thought. A classification according to subject-matter—such as groups of illnesses, organ systems, or technical procedures—would only have accomplished our purpose very incompletely. Unlike the situation in the rest of medical science, it happens in psychosomatic medicine that such things as the initial point of view, the method of research and interpretation of results, differ so very widely amongst different authors that the mere placing of a work under this or that subject-heading would tell the reader comparatively little about its contents. This difficulty is particularly great in German; for the tendency to try and establish individual theories, the urge to wrestle with fundamental questions and as far as possible range them under this or that philosophical system, the preference for using a complicated language (often with its own terminology) and a striving after originality—these elements not only promote the development of a great variety of concepts, but make reading much harder for the outsider. We therefore decided to carry out, as well as the classification according to subject-matter, an additional classification according to the different trends, schools and methods of working, although this entailed two disadvantages:

(1) Very few authors take the trouble to specify precisely their basic point of view, the field of their investigation, and their method. Thus the categorization of works under this or that trend, only reflects our own interpretation, which no doubt is frequently disputable.

(2) In many cases the points of view cut across this classification, in such a way as to permit only an incomplete, and frequently one-sided, characterization of the factual content.

Nevertheless it seems preferable to attempt this, rather than leave the reader completely in the dark concerning the manner and the standpoint of investigation.

Accordingly the survey will be divided into three parts:

(1) In the first part we shall try to offer a general view of the different trends, their differences and points of contact. By this means we hope to help the reader find his bearings amid the confusing landscape of our psychosomatic literature, and

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also to provide some general information about the situation of psychosomatic medicine in Germany.

(2) Secondly the reader will find a classification of the literature—both according to subject-matter, and also as to their basic standpoint and tendency as set out in (1).

(3) Thirdly there follows a Bibliography, in alphabetical order.

I

THE DIFFERENT BASIC STANDPOINTS AND TRENDS

(1) *Symbolic meaning and the science of expression*

Ever since the early days of psychoanalysis when an insight was gained into the symbolic character of neurotic slips, hysterical symptoms, and above all, of dreams, attempts have continually been made to treat somatic symptoms of organic diseases from this point of view. Particularly in Germany, there were already precursors of this view in the first half of the nineteenth century. The idea of the significance of bodily phenomena as an expression of psychic activity enjoyed particular popularity amongst the German Romantics. It can therefore be understood how the seeds of psycho-analysis fell on particularly favourable ground. GRODDECK was certainly the first to grasp these possibilities and carry them almost to excess. His influence on the first researchers in the sphere of psychosomatic medicine in Germany after World War I is unmistakable.

In addition to the psychoanalytic doctrine of symbolism, there is also an interpretation of expression, which is to be conceived partly as a continuation of the attempts of the German Romantics. It extends from an interest in graphology, and mimics, to an actual attempt to *comprehend* bodily symptoms as a form of "organ dialect" and ultimately to treat the whole body as an expression of psychic content. This tendency is only partly in touch with the doctrine of symbolism centred round psychoanalysis.

A common element in both tendencies is that they do not attempt to support their hypotheses and conclusions with any rational proofs by means of experimental or statistical investigations; rather they are both convinced that the only adequate method lies in an intuitive comprehension of the expression, e.g. of the symbolism of bodily symptoms.

(2) *The anthropological approach*

The science of expression is concerned with the hidden or locked-away meaning of this or that symptom, as a symbol. The question of meaning becomes far more pretentious when it is taken to include the meaning of human life in general. In this form the question of meaning is posed more or less openly and fundamentally, in the so-called "anthropological" tendency. This occurs in the most evident and radical manner in the case of the Nestor of German psychosomatic medicine, who has only recently died, VIKTOR V. WEIZSÄCKER, who has advanced the thesis that the significance of illness is to guide the patient towards the meaning of his own life. But if every illness has its specific meaning in the life history of the patient, then one can understand why it is that for V. WEIZSÄCKER, biography offers the key to the understanding of illnesses.

The attempt to approach the question of meaning in this form conceals many dangers. On the one hand it may lead to standards and values being introduced

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into psychosomatic medicine, of which the appropriateness in that field seems highly questionable. In the last resort it leads certain authors to maintain that the sick person belongs to an élite, in the face of which the ranks of healthy people seem inferior and obtuse, since they lack the sensitivity to grasp the meaning of a life that has succeeded or miscarried. Another danger lies in the dogmatic attitude that may lurk beneath the terminology of an anthropological medicine. The word serves as a kind of healing token, under whose shadow the community of the elect assembles, or as a means to strike terror into the unbelievers. It is therefore beneficial that v. GEBSATTEL, one of the leading psychiatrists of the anthropological school in Germany, has emphasized that the present time is not yet ripe either for a medical or indeed for a philosophical anthropology. After what has been said, it is by no means astonishing that once again very many different kinds of tendencies have laid claim to the title of an anthropological medicine. The common element amongst all these is frequently no more than the emphasis laid on biological methods, by which one tries to place "Man" in the very centre of attention. It is obvious that many misunderstandings will arise here, and that many very different kinds of definition of the concept "anthropological" will be found. Hence, for purposes of classification, we shall put together under the heading "anthropological tendency" only such works as pose the question of meaning in a metaphysical connection, as happens in an exemplary fashion in the case of v. WEIZSÄCKER.

(3) *Existential analysis*

This approach originated in Switzerland, where it was founded by the psychiatrist and pupil of FREUD, BINSWANGER. Since it is also gaining supporters in Germany in increasing numbers, it must be mentioned here. It is difficult to define this viewpoint with real precision and to distinguish it from the anthropological approach. There is much overlap, and much in common between the two approaches. Perhaps one can say that, to a special degree, existential analysis is founded on a phenomenological method, by which it tries to separate off certain specific human forms of behaviour in healthy people and in sick people. It is centred round the phenomenological works of HUSSERL and the philosophy of HEIDEGGER. Once again, it shares with the anthropological approach an often harsh rejection of scientific medicine, with its experimental methods.

(4) *Psychoanalytically oriented trends*

Under this heading we shall place works that have been accomplished by methods that are predominantly psychoanalytical, although here too there is much overlap, and contact with other tendencies. This is chiefly the case with the interpretation of expression and symbolism; it may be connected with the fact that the division made by ALEXANDER, between Conversion (as an expressive and symbolic event) and Organ Neurosis (as a non-expressive internal process, determined more in a vegetative-nervous manner) has not been so universally accepted here as in the U.S.A.

Here there are chiefly two schools that ought to be mentioned; both endeavour to replace the original concept of primitive symbolic interpretation with differentiated ideas.

(a) After World War II, SCHULTZ-HENCKE and his Berlin Institute came to the fore on account of his attempt at "amalgamating" the deep-psychological tendencies

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of FREUD, ADLER, and JUNG. His theory—which, in spite of its thoroughgoing application of psychoanalytic thought-processes, gave rise to much hesitation among orthodox analytical circles—is enjoying a very wide diffusion in Germany. In connection with the pathogenetic problem of psychosomatic medicine it advances (*inter alia*) the following hypothesis: owing to some misintegrational flaw (e.g. displacement) it may be possible for a part of the originally fully-conscious motive-experience to go astray, and become a "splinter-motive" which can then manifest itself as a neurotic symptom or even as a somatic clinical picture.

(b) The working-group around MITSCHERLICH in Heidelberg is founded more than other groups on the classical methods of psychoanalysis. To a certain extent, therefore, it has close connections with analytic schools in England and the U.S.A. MITSCHERLICH's concept of pathogenesis starts from the principle that one must distinguish two different psychomatic processes:

(i) Acute intercurrent illnesses in the case of relatively un-neurotic people, which are occasioned by a structural or impulse-conflict. The purpose of such illnesses must be to solve the conflict, and bring about a change in personality and a genuine personal mastery of one's life. (Here can be seen MITSCHERLICH's connection with the anthropological school, as represented by V. WEIZSÄCKER, whose successor he is.)

(ii) In the case of chronic illnesses, preceded (especially in early life) by gross neurotic maladjustments, the disappearance of the neurotic phenomena will be accompanied by the emergence of somatic symptoms. The symptom is to be interpreted here as the formation of a compromise between the Super-Ego and the Id. In this connection MITSCHERLICH speaks of a "dual-phased displacement," since in the first phase the neurotic symptom-formation was incapable of adequately mastering the conflict or the crisis, and hence it was only in the second phase, by shifting over to the dynamics of bodily processes, that the displacement-process was able to reach its conclusion.

(5) *Kretschmer's constitutional biology*

Whilst in general German psychiatrists adopted an attitude of expectancy or scepticism towards psychosomatic medicine, the Tübingen psychiatrist KRETSCHMER, together with a few similarly minded colleagues, has for many years been carrying out decisive investigations concerning the connection between bodily structure and bodily functions on the one hand, and psychic functions on the other. He takes his stand by a psychotherapy and theory of neurosis which pays much greater attention to general biological factors. In addition to the connection between constitution and character, he pays particular attention to the discrepancy between bodily and psychic maturity as a pathogenetic principle.

(6) *The "organismic" approach of* SCHULTZ

Connected with these ideas is the school of thought represented by the Berlin psychiatrist SCHULTZ. He has developed a system of "autogenous training"—a method of concentrational self-relaxation which is related to the practices of Yoga. He stresses an "organismic" aspect in the approach to neuroses and psychosomatic illnesses.

(7) *The psycho-physiological approach*

Under this heading we shall group works which, in spite of differences in standpoint and methods, do share a common striving towards an experimental solution of

psychosomatic problems. They all rest on the basic idea, which was stressed particularly by G. v. BERGMANN, that anatomical organic injuries can be occasioned by functional dislocations. Thus they seek to investigate the connections that exist between the emotions and effects, and such "functional disturbances." But they differ from each other, nevertheless, on account of their different interpretations of what these connections are like.

(a) Many works are content to give a simple record of functional alterations as a result of (e.g. hypnotically produced) affects.

(b) Others attempt to advance to a kind of "physiology of the emotions" and to discover definite regular rules of co-ordination. In the process, many start out from the classical ideas of a psychophysical parallelism, or of a reciprocal causal action between the psychic and the physical. Some speak of a "correlate of simultaneity;" others, of a "psychogeny" of somatic processes—something that is chiefly to be met with in the work of investigators who describe themselves as supporters of SELYE's theory of Stress. Other investigators attempt to advance by means of totalist (holistic) and teleological conceptions. The impetus for this is to be found in the works of CANNON concerning emergency-functions, and the investigations of W. R. HESS concerning teleological arrangement in the growth of living functional systems, and also in the theories of behaviour put forward by TINBERGEN and LORENZ. Publications of this group concern themselves with patterns of integration for inborn or acquired functional systems, or attempt to investigate "moods" as psychophysical "states of preparation."

(c) Lastly there belong here also those works which investigate by means of statistics the relationship between different vocational groups or life-situations and psychosomatic conditions.

(8) *Works of a more casuistic character*

Here we group together a fairly large series of works which cannot be assigned to any particular line of approach. They record psychosomatic observations in definite situations (anxiety, hunger, release), or else define their position (on the basis of a purely descriptive casuistic method) towards the psychosomatic aspects of definite clinical pictures. Non-analytic biographies are especially numerous. The fact that there are so many works of this kind is certainly not due solely to an inclination in German medicine towards casuistic methods. It is also to be accounted for by the serious state of affairs, which is such that, for material reasons, only very few doctors who are interested in psychosomatic matters have undergone a specialized training.

(9) *Socio-psychological approach*

In imitation of many examples in Great Britain and the U.S.A., the points of view of social psychology are entering into psychosomatic ideas in Germany also; although, only thirty years ago, V. v. WEIZSÄCKER put forward a plea that more attention should be paid to different social factors in the widest sense.

(10) *Central nervous system and psychic processes*

A number of investigators have concerned themselves with the relations between neurophysiological processes in the brain and psychic events; the corresponding literature is listed under this head.

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(11) *Tests and delimitations of the personality-characteristics of sick people*

(12) *Works of a purely theoretical nature*

(a) Amongst German writings these are comparatively numerous. They include essays which discuss the body-mind problem. Others are concerned with the extent of the province of psychosomatic processes, or with pathogenetic concepts. In addition works are listed here which offer some criticism of certain ideas and methods in psychosomatic medicine, in so far as they can be described as positive contributions.

(b) Polemics: There are also criticisms, which condemn psychosomatic medicine as a whole. They are rarely based on personal experience, but usually consist of violent polemics, for the most part delivered in a thoroughly dogmatic manner. But since they produce definite arguments and reflect characteristic attitudes which are continually recurring, we thought it was proper to include them separately as item 12(b) in our survey.

II

*Classification by Tendencies**

(1) Symbolic meaning and the science of expression: 1, 9, 45R, 109R, 111, 138, 161, 165R, 244, 245, 246.

(2) The anthropological tendency: 20, 27, 28, 29, 56, 86, 87, 88, 89, 97, 104, 105, 106R, 107, 146, 160, 161, 162, 163, 193, 194, 195, 213, 244, 245, 246, 254, 256.

(3) Existential analysis: 125.

(4) The tendencies centred round psychoanalysis:

(a) Berlin School (SCHULTZ-HENCKE): 1, 2, 13, 30, 31, 32, 33, 40, 41, 74, 84, 123, 124, 135, 168, 184, 185, 199R, 207, 208, 209, 210, 211, 212, 230.

(b) Heidelberg School (MITSCHERLICH): 11, 18, 19, 21, 92, 141, 142R, 143R, 144, 150, 179, 180, 214, 215, 219, 223, 224, 231, 255, 257.

(5) KRETSCHMER's constitutional biology: 70, 71, 72, 73, 77, 127, 130, 174, 175, 227, 228, 250.

(6) The "organismic" tendency of J. H. SCHULTZ: 182, 183, 196, 197R, 198.

(7) The psycho-physiological tendency:

(a) Simple registration: 8, 46, 66, 100, 113, 114, 116, 117, 118, 119, 131, 132, 151, 154, 170, 171, 186, 188, 203, 205, 241.

(b) Investigations into the physiology of emotions: 28, 29, 42, 44, 50, 158, 159, 217, 229, 233, 234, 235.

(c) Statistical investigations: 22, 34, 35, 253.

(8) Works of a more casuistic character:

(a) Anxiety state: 153R, 222.

(b) Hunger: 6, 44, 54, 55, 155, 190.

(c) State of release: 157, 192.

(d) non-analytic biographies and observations: 5, 14, 15, 16, 17, 23, 37, 49, 51, 52, 53, 57, 59, 60, 64, 65, 69, 70, 71, 72, 73, 76, 78, 79, 80, 81, 94, 95, 98, 102, 103, 108, 110, 112, 115, 121, 122, 126, 134, 136, 137, 145, 147, 148, 152, 169, 172, 173, 176, 177, 187, 189, 191, 200, 201, 202, 204, 206, 218, 220, 238, 239, 240, 248, 249, 251, 252, 259.

* Works marked with an "R" are surveys of a limited field, usually containing additional bibliographical references.

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(9) The points of view of social psychology: 12, 51, 75, 83, 89, 156.
(10) The central nervous system and psychic processes: 7, 25, 26, 36, 38, 39, 69, 90, 128R, 201, 222.
(11) Tests and delimitations of the personality characteristics of sick people: 3, 4, 48, 58, 59, 61, 62, 63, 120, 129, 139, 140, 164, 178, 225, 226, 247, 258.
(12) (a) Works of a purely theoretical nature: 24, 27, 43, 67, 68, 82, 91, 93, 96, 97, 101, 106R, 142, 143, 149, 159, 166R, 181R, 189R, 216, 221, 232, 236, 237.
(b) Polemic: 133, 167, 242, 243R.

Classification by Subject-matter

(A) *Circulatory organs*

(1) Hypertension: 203, 231, 249, 254, 256.
(2) Circulatory regulation: 2R, 8, 51, 118, 132, 151, 170, 171R, 188, 202, 239.
(3) Vasolability: 2R, 13, 76, 160, 250.
(4) *Angina pectoris:* 80, 81, 83, 189.
(5) ECG-changes: 119, 131, 170, 171R.
(6) Paroxysmal tachycardia: 168, 239, 257.

(B) *Digestive organs*

(1) Gastric function: 208R, 233.
(2) Peptic ulcer: 12, 22, 57, 58, 59, 60, 61, 62, 63, 64, 74, 81, 83, 199, 207, 208R, 218, 233, 253.
(3) Aerophagy: 78.
(4) Cardiospasm: 112.
(5) Colitis: 74, 183, 208R.
(6) Jaundice: 72.
(7) Gall-bladder disease: 83.
(8) Constipation: 169.

(C) *Respiratory organs*

(1) General: 209R, 240R.
(2) Nervous breathing-syndrome: 28, 29.
(3) Bronchial asthma: 3, 19, 21, 94, 123, 164, 176, 177, 209R, 210, 211, 230, 204R.

(D) *Kidney and urinary tract*

(1) Functioning of kidneys and maintenance of urine: 1R, 118, 141, 204R.
(2) Enuresis: 1R, 105R, 246.

(E) *Endocrine organs and metabolism*

(1) Basal metabolism: 46, 47R, 50, 66, 100, 116.
(2) Obesity: 223.
(3) Anorexia: 40, 125, 130, 145, 199, 219, 259.
(4) Hyperthermia: 17, 159.
(5) *Diabetes mellitus*: 32, 33, 85, 152, 258.
(6) *Ovary*: 36, 95, 22.
(7) *Morbus Cushing*: 69.

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(8) Thyroid gland: 48, 75, 83.
(9) Tetany: 49.
(10) Hypophysis: 141, 205.
(11) Suprarenal gland: 115, 205.
(12) Testes: 95, 222.

(F) *Musculo-sceletal system*

(1) General: 30R, 45, 111, 122, 184R, 185.
(2) Rheumatism: 30R, 161, 185.
(3) Vertebral column: 30R, 98, 184R, 185, 227, 228.

(G) *Nervous system*

(1) General: 127, 129, 191, 192, 206, 225.
(2) Epilepsy: 18, 92.
(3) Diencephalon: 25, 128, 186, 187.
(4) Vegetative nervous system: 77, 132, 160, 215, 222.
(5) Cerebral injuries: 38, 39, 128R.
(6) Extrapyramidal disturbances: 20, 229.
(7) Injuries to spinal cord: 7, 224.
(8) Multiple sclerosis: 26, 226, 251, 252.
(9) Vertigo: 160, 162, 212.
(10) Headaches: 193, 194, 195.

(H) *Sensory organs*

(1) Eyes: 14, 37, 71, 200, 201, 238.

(J) *Skin*

(1) General: 15, 16, 147, 172.
(2) Urticaria: 70, 73, 246.
(3) Dermatoses: 15, 16, 124.
(4) Sexual organs: 84, 104, 135.
(5) Hair: 79, 110.

(K) *Gynaecology*

(1) General: 9, 52, 56, 102, 103, 135, 136, 167, 174R, 175, 182, 220.
(2) Obstetrics: 10, 53, 65.
(3) *Hyperemesis gravidarum*: 173, 174.
(4) Pseudocyesis: 108.
(5) Menstrual disorders: 121, 126, 134, 136, 137, 174.

(L) *Infectious diseases*

(1) Tuberculosis: 5, 86, 87, 88R, 89, 120, 139, 140, 146, 178, 247, 248.
(2) Diphtheria: 90.

(M) *Blood, malignancy*

(1) Blood formation: 113, 117, 132, 154, 241.
(2) Blood chemistry: 114, 117.

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(3) Pernicious anaemia: 23.
(4) Malignant tumours: 163.

(N) *Paediatrics*

11, 41, 122, 148.

(O) *General syndromes*

(1) Hunger: 6, 44, 54, 55, 155, 190.
(2) Nausea: 44, 50, 229, 232, 234R, 235, 236.
(3) Anxiety: 131, 153R, 218.
(4) Sleep: 42.

(P) *Allergy*

21, 70, 73, 88, 94, 150, 213R, 250.

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REPORT OF MEETINGS

SOCIÉTÉ FRANÇAISE DE MÉDECINE PSYCHOSOMATIQUE

Compte rendu de la séance du 28 février 1957

MM. SAPIR, LABHARD et SHENTOUB—Essai d'étude psychosomatique de l'hypertension artérielle.

Les auteurs ont divisé les hypertendus en deux groupes, le premier présentant des troubles psychologiques et nerveux, le deuxième exempt apparemment de ces troubles. Le tri a été fait par des médecins—somaticiens, puis chacun de ces malades a été soumis à un examen psychiatrique approfondi. Des tests projectifs ont été pratiqués (T.A.T. et RORSCHACH). Les quarante malades des deux groupes sont suivis depuis plusieurs années à la consultation et la plupart ont subi au moins une hospitalisation.

Il n'y a pas de parallélisme entre les modifications du comportement et les formes cliniques somatiques. Mais le plupart des hypertendus présentent des réactions caractérielles différentes de celles du nevrosé classique.

De même l'étude de l'histoire de la maladie n'a permis d'établir aucune corrélation entre l'évolution et la structure de la personnalité.

MM. GILBERT-DREYFUS et R. HELD—À propos de quelques cas d'obésité—quelque réflexions cliniques.

Les auteurs critiquent les notions d'obésité exogène et endogène. Après un rappel de l'expérimentation classique, ils présentent cinq observations psychosomatiques. Troubles de la faim ou troubles métaboliques sont en rapport avec des conflits.

Les significations psycho-affectives inconscientes de l'obésité, les réactions du patient à la maladie, les anomalies sexuelles (frigidité quasi-constant), le cercle vicieux psychosomatique (angoisse, polyphagie, obésité-culpabilité-angoisse) sont précisés et discutés.

M. H. P. KLOTZ—À propos des obésités d'origine psychosomatique.

L'auteur rapporte un grand nombre d'obésités à déclenchement psychogène. Elles succèdent tantôt à un choc affectif violent et unique, tantôt à une série de chocs répétés ou à une situation conflictuelle, tantôt à un surmenage, parfois professionnel du système nerveux central.

Certes, certaines de ces obésités s'accompagnent d'une polyphagie anxieuse et leur mécanisme de production est ainsi assez simple.

Mais la plupart ne s'accompagnent pas de cette polyphagie et l'on doit faire intervenir pour expliquer l'obésité, une perturbation fonctionnelle hypothalamique modifiant profondément les équilibres métaboliques antérieurs. En faveur de cette conception, l'auteur montre, dans ces divers types d'obésité psychosomatique, des témoins indiscutables de perturbation diencéphalique, en particulier, une hypersécrétion d'antidiurétine hypophysaire postérieure et de cortrophine hypophysaire antérieure.

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Compte rendue de la séance du 7 avril 1957, à Paris

Études sur les méthodes psychologiques en analgésie obstétricale

Le 7 Avril s'est tenue à la Faculté de Médecine de Paris une journée d'étude sur les méthodes psychologiques en analgésie obstétricale, organisée par la Société Française de Médecine Psychosomatique et placée sous la présidence du Doyen L. Binet. M. F. Moutier qui présida la première séance a, dans son introduction, rendu un émouvant hommage au docteur Lamaze, qui a introduit en France la méthode psychoprophylactique de préparation à l'accouchement: "L'apostolat est rare aujourd'hui, mais vraiment Lamaze a été, dans toute l'étendue du mot, dans tout son élan de charité et d'intelligence, un apêtre".

J. Ajuriaguerra (Paris), dans le rapport d'introduction a magistralement posé le problème de la douleur, tel qu'il se présente de nos jours. Après avoir affirmé la spécificité des récepteurs de la douleur, le rapporteur expose les connaissances actuelles du système sympathique dans la conduction des sensations viscérales. Il discute le rôle du thalamus et de la substance réticulée dans le cheminement ascendant des sensations douloureuses. Au-delà du thalamus l'auteur souligne que "le devenir des afférences de la douleur est problématique". Il expose ensuite quelques données de physiologie expérimentale concernant le problème du seuil de la douleur, de la sommation temporelle et spatiale. Il note les variations des seuils lors de l'application simultanée d'autres stimuli (douloureux ou non) aboutissant à des élévations importantes des seuils.

Sur le plan clinique, l'auteur rappelle que certaines lésions des fibres nerveuses entraînent la disparition des sensations douloureuses, sans diminution des sensibilités tactile et profonde. Au contraire, d'autres lèsions nerveuses, notamment thalamique, provoquent des algies. De nombreux auteurs ont voulu faire du thalamus le centre intégrateur spécifique de la douleur, mais les faits physiologiques et neuro-chirurgicaux (notamment les lobotomies) montrent le rôle indiscutable du cortex dans l'intégration de la douleur.

Après l'exposé des faits, l'auteur tente l'analyse de la sensation *douleur*. Il montre tout d'abord que cette sensation ne se conçoit pas sans son côté "centripète" simultané: autant qu'une perception, la douleur est une réaction. . . . Cette réaction dépend de chaque sujet, de sa personnalité, de son psychologie. L'auteur évoque l'anesthésie hystérique, la suggestion verbale et non verbale. Il montre aussi l'aspect social de la douleur, c'est à dire l'interrelation entre le sujet qui souffre et l'entourage. Tous ces facteurs font partie de l'étude de la douleur qui apparaît ainsi comme un phénomène intersubjectif.

L.Chertok (Paris) expose ensuite l'évolution des idées sur l'analgésie psychologique en obstétrique, ouvrant ainsi le débat sur les méthodes actuelles. Il rappelle les deux méthodes principales les plus courantes: la méthode de Read (d'accouchement sans crainte) et la méthode de Velvovski (couramment appliquée en Union Soviétique et universellement connue sous le nom "d'accouchement sans douleur") et souligne le peu de différences techniques existant entre elles. L'auteur explique qu'aucune théorie valable ne peut encore éclairer de façon certaine le processus qui provoque l'analgésie. Il s'agit d'un problème psychophysiologique complexe et mal connu.

Dans la seconde partie de son exposé, l'auteur traite des rapports de l'analgésie

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psychologique et des phénomènes d'hypnose. Il rappelle que l'hypnose produit une analgésie d'origine verbale, qui d'ailleurs a été utilisée en obstétrique dès la fin du dix-neuvième siècle en France, puis en Allemagne et en Autriche vers 1920. Ce sont les psychiatres russes PLATONOV et VELVOSKI qui ont repris ces recherches. Ces auteurs ont été conduits à nier le caractère inéluctable de la douleur au cours de l'accouchement et, d'autre part, à concevoir l'idée d'une analgésie verbale, en accord avec la théorie pavlovienne. Mais l'hypnose comme élément d'analgésie obstétricale n'a pas reçu de développements importants et VELVOSKI a mis au point une autre méthode accessible à chaque médecin: c'est la méthode psycho-prophylactique.

Ainsi la méthode psycho-prophylactique a pris la place de la méthode hypno-suggestive, pour des raisons pratiques, bien que sa base théorique soit encore moins ferme. Pour VELVOVSKI, la douleur de l'accouchement n'est pas un phénomène physiologique, mais le résultat d'un conditionnement social. Le but est de rompre la douleur réflexo-conditionnée; il ne s'agit pas "d'inhiber la douleur, mais d'activer le cortex". Cependant, même en U.R.S.S., les auteurs ne s'accordent pas sur ces interprétations théoriques: beaucoup croient au caractère physiologique de la douleur de l'accouchement et admettent le rôle de la suggestion, donc de l'inhibition, dans l'analgésie psychologique. Entre les deux tendances aucune preuve experimentale n'a pu encore être apportée.

Dans la troisième partie de son exposé CHERTOK pose le problème des types nerveux. Il pense que la classification des gestantes selon des types, permettant de prévoir quelle sera leur réponse à l'anal gésie verbale est actuellement un leurre. Elle ne permet pas d'expliquer les réactions individuelles au cours de l'accouchement. D'autres recherches, établies à l'aide de tests de personnalité, sont en cours.

En conclusion, l'auteur souligne l'immense intérêt pratique que présentent les méthodes d'analgésie psychologique et le champ d'investigation théorique qu'elles ont ouvert aux chercheurs, posant le problème des interrelations psychophysiologiques.

"Cinq ans d'experimentation de la méthode psycho-prophylactique d'accouchement sans douleur" retracent l'immense travail réalisé à Paris par le Docteur F. LAMAZE. Le rapport était lu par P. VELLAY (Paris) qui fut son collaborateur dès les premiers jours. Le rapporteur estime que ces méthodes ont toutes eu le mérite d'améliorer la "condition" des femmes en couche, mais qu'elles sont aujourd'hui dépassées par la méthode psycho-prophylactique qui constitue actuellement la méthode la plus sûre, la plus efficace.

Après un rappel historique, le rapporteur expose le bilan d'ensemble de l'accouchement sans douleur.

Dans l'appréciation des résultats, il distingue trois catégories d'accouchement:

(1) Les femmes pour lesquelles les conditions physiques, psychiques et obstétricales sont bonnes: dans cette catégorie on obtient 90 pour cent d'indolorisation absolue.

(2) Les femmes dont l'accouchement est à la limite de la dystocie, que ce soit pour des raisons physiques, psychiques ou obstétricales: dans cette catégorie on peut encore obtenir de bons résultats sans médications,

(3) Les femmes dont l'accouchement est dystocique et où l'apport médicamenteux est indispensable, quoique à faibles doses.

notions qui lui semblent capitales. Il insiste ainsi sur la rigueur absolue qu'il faut observer dans les diverses phases de la préparation, sur la notion d'équipe, sur le rôle

de la famille et surtout du mari. Il rappelle que la femme doit être soutenue dans son effort par un climat d'enthousiasme et de conviction, ainsi que par une ambiance matérielle satisfaisante (locaux, salle de travail). Il expose ensuite les investigations qui portent sur le "passé pathologique" et sur le "climat psychologique" de chaque femme, sur l'éducation qu'elle a reçue dans sa jeunesse, ses conditions de vie actuelles et, bien entendu, sur l'état physique actuel. C'est l'étude détaillée de ces diverses données qui permet de "détérminer dans quelle direction devra porter l'effort de l'éducateur". Le rapporteur reconnaît d'ailleurs combien la prévision individuelle du résultat est aléatoire. Il relate ensuite les données recueillies aurpès des femmes ayant suivi la préparation à l'accouchement sans douleur: motivations les ayant poussé à pratiquer cette méthode, impressions sur la préparation, l'équipe des accoucheurs et l'accouchement lui-même.

Dans la quatrième partie de l'exposé il insiste sur le haut degré de conscience atteint par la femme au cours de l'accouchement, puis sur la possibilité d'appliquer le forceps sans anesthésie.

F. Lepage (rapporteur) et G. Langevin-Droguet (Paris), quant à eux, appliquent depuis 1949 une méthode qui dérive de celle de Read. Les femmes reçoivent une préparation psychologique (explications physiologiques sur la gestation et l'accouchement, mise en confiance) et physique (mouvements respiratoires, mouvements abdominaux, relaxation). La partie prépondérante est la préparation psychologique. Quatre-vingts pour cent des femmes ont tiré un bénéfice certain de la méthode, mais les auteurs pensent que la suppression totale de la douleur n'est pas le test du succès; il leur suffit que les sensations au cours de l'accouchement soient supportables et permettent à la femme de "garder le contrôle". C'est pourquoi les auteurs préfèrent le terme d'accouchement sans crainte à celui d'accouchement sans douleur. Il convient de souligner que cette méthode a permis d'une part de diminuer l'emploi d'anesthésiques ou d'analgésiques, d'autre part d'augmenter le nombre de nouveaux-nés ayant crié dès leur naissance. Par contre, il n'y a aucune modification significative du nombre des complications obstétricales.

Le Dr. G. D. Read (Londres) a alors exposé ses idées sur la douleur de l'accouchement, idées sur lesquelles il a fondé sa méthode. Il rattache les douleurs des parturientes à une triade: crainte, tension, douleur. "Si la douleur", dit-il, "est supprimée par l'anesthésie, l'enfant et la mère courent des risques. Supprimer la tension ne suffit pas . . . Si la crainte est enlevée le résultat est bien meilleur. Si la tension et la crainte sont supprimées, dans environ 90 pour cent des cas, la douleur est supprimée." L'auteur indique qu'il obtient la suppression de la tension par la relaxation et celle de la crainte par l'éducation. La méthode ainsi apparaît comme une technique dont la justification est fournie par les excellents résultats statistiques.

M. Mayer et A. Morali-Daninos (Paris) emploient à l'hôpital Saint-Antoine une préparation à l'accouchement comportant également conférences (préparation psychologique) et exercices (préparation somatique). Cette "aide psychosomatique" qui s'étend de la période prénatale à la période post-natale permet, disent les auteurs, de réaliser un "accouchement naturel". Cependant, une défaillance étant fréquente au moment de l'expulsion, ils ont souvent recours à des méthodes médicamenteuses dans les derniers moments de l'accouchement.

Au cours de la séance de l'après-midi, placée successivement sous la présidence de MM. Malcovati et Geisendorf, une discussion animée s'est déroulée à propos du

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transfert affectif que la parturiente établit sur la personne de l'accoucheur et que les psychiâtres et M. READ invoquent pour expliquer les bons résultats de l'analgésie verbale. VELLAY déclare que ce transfert est gênant car il oblige l'accoucheur à être toujours présent et qu'il peut se faire sur toute l'équipe. MORALI parle aussi de transfert sur l'équipe, notion réfutée par JIMENEZ (Madrid) et COTTEEL (Lille).

C'est également au cours de la discussion qu' AJURIAGUERRA a montré de façon très concrète que les relations entre les hommes ne sauraient être ramenées uniquement aux échanges par la parole (deuxième système de signalisation de PAVLOV), en réalité, bien d'autres données sensorielles président à ces échanges et là est la base de la psychologie, science des relations interpersonnelles.

Après la discussion des rapports, de nombreuses communications ont été présentées:

H. ZAIDMAN (Paris) applique depuis deux ans la méthode de VELVOVSKI dans des centres de santé de la région parisienne. Cet auteur évalue à 20 pour cent le nombre des échecs (sur 192 cas). Ces échecs ont souvent été provoqués par l'ambiance psycho-hygiénique des centres d'accouchement non adaptés aux idées nouvelles.

B. THIS (Paris) a présenté une "introduction à la psychopathologie obstétricale" d'inspiration psychoanalytique sous la forme du "Mythe d'Alcmène". À travers le récit d'Ovide relatant l'accouchement difficile de l'épouse d'Amphitryon, il analyse les racines inconscientes de la douleur de l'accouchement.

R. HELD (Paris) apporte un autre point de vue psychoanalytique sur les rapports entre la nervose et la grossesse. Il pense que la parfaite compréhension de l'analgésie verbale comporte "une référence obligatoire à la dimension psychoanalytique". Ceci; parce que la peur est, d'un avis unanime, le point de départ de la douleur. Or la peur est le fruit des motivations inconscientes. Il a donc semblé intéressant à l'auteur d'analyser les motivations de la peur au cours de la grossesse chez des névrosées: ces femmes, anormales certes, révèlent des processus qui jouent certainement aussi chez des normales. Il souligne l'importance du transfert dans la liquidation de la peu grâce à la perte acceptée de la vigilance, et extrapole cette interprétation à l'accouchement sans douleur. Selon lui, ce transfert seul permet à la longue la perte de vigilance, l'abandon au cours de l'aventure existentielle de l'accouchement.

A. ECONOMIDES et H. VERMOREL (Lyon) présentent un bilan portant sur 400 accouchements selon la méthode de VELVOVSKI qu'ils ont vu appliquer par LAMAZE et VELLAY. Ils étudient l'état nerveux des femmes au début de la préparation, partant, dans certains cas, d'une nevrose réactionnelle. Au cours de la préparation, ces symptômes disparaissent progressivement. Les auteurs soulignent ensuite les difficultés que comportent les applications statistiques des résultats: l'évaluation "objective" de la douleur étant impossible. Sur les dires des parturientes ils pensent que 50 pour cent de leurs patientes n'ent ressenti *aucune* douleur. Le nombre de véritables échecs est très modeste (5 pour cent). Les auteurs terminent leur communication en reconnaissant les difficultés que rencontre une interprétation théorique des résultats obtenus par la méthode.

J. SEARRA-DINIS (Lisbonne) discute, dans une perspective pavlovienne, les rapports de la douleur et de l'accouchement, de la suggestion et du langage. . . . Il reconnaît les insuffisances actuelles des tentatives d'explication. Mais l'etude pavlovienne de ces problèmes élargie et enrichie par les travaux physiologiques (tels que ceux concernant la substance réticulée) doit conduire les recherches dans la voie la plus fructueuse.

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J. ROUX (Genève) dans le service du Professeur DE WATTEVILLE a tenté d'établir un rapport entre la réussite de l'accouchement sans douleur et le niveau intellectuel des parturientes. La méthode de préparation à l'accouchement est celle de VELVOVSKI. Le niveau intellectuel est apprécié à l'aide de quatre tests psychologiques. Les auteurs ont observé que quand les conditions obstétricales sont bonnes l'accouchement a d'autant plus de chances de réussir que le niveau est meilleur.

A. BOCCI et L. DAVITTI (Clinique universitaire de Turin, Professeur G. DELLEPIANE) constatent une relation inversement proportionnelle entre la durée supposée de la gestation et le comportement. Le nombre de parturientes dont le comportement est médiocre pendant le travail double lorsque survient un retard important par rapport à la date prévue de l'accouchement. La préparation psycho-physique modifie cet état de choses du fait que la parturiente comprend les possibilités d'erreur de date, qu'elle attend la délivrance sans anxiété. Comparaison entre 700 cas préparés et 4046 cas non préparés.

G. CHIAUDANO et G. PINOLI (service du Professeur DELLEPIANE) essaient partant de la réfléxologie pavlovienne d'étudier la méthodologie audio-visuelle pour le conditionnement de la gestante. Ils commentent les résultats obtenus par l'emploi du film et du dessin animé.

I. TERZI (travail effectué dans le service du Professeur DELLEPIANE) envisage le comportement psycho-moteur de la gestante comme un tout réflétant la personnalité, aussi bien à son niveau superficiel-sensoriel, qu'au niveau plus profond de ses pulsions. Il analyse sur un plan psychosomatique les trois aspects de la conduite motrice de la gestante: l'aspect expressif, l'exécutif, l'impressif. C'est ce dernier aspect qui dérive de l'état intime et révèle les particularités psychologiques de la personnalité.

L'auteur se livre à une analyse psychosomatique poussée des diverses étapes de l'accouchement selon le type psychologique de la gestante.

A. NOTTER (Lyon) rapporte les résultats d'une intéressante étude de pneumospirométrie poursuivie dans le service du Professeur PIGEAUD. La méthode employée par les accoucheurs de ce service est inspirée tant de la méthode de VELVOVSKI que de celle de READ. L'auteur a soumis à des tests spirométriques 1243 femmes qui suivaient la préparation à l'accouchement. Il peut ainsi chiffrer les performances respiratoires (capacité vitale, contrôle des rythmes accélérés, temps d'apnée) au cours de l'évolution de la grossesse. Il constate que, statistiquement, les femmes ayant accompli avec succès les épreuves spirométriques accouchaient mieux.

W. GEISENDORF (Genève) fait part des progrès de l'accouchement sans douleur dans cette ville. Il retrace les difficultés initiales, créées surtout par le corps médical. Là encore, c'est le public qui en fait a imposé cette méthode. En dehors de toute considération théorique, l'auteur affirme l'immense bénéfice pratique accordé aux parturientes, bénéfice qui, à lui seul, justifie tous les efforts réalisés pour développer la méthode.

La Société Gynécologique Espagnole avait délégué A. HERNANDEZ JIMENEZ (Madrid) à cette journée d'études. L'auteur rend hommage au Dr. LAMAZE auprès duquel il a acquis en 1953 la pratique de la méthode appliquée actuellement à Madrid. Là aussi de nombreuses difficultés, tant de la part des accoucheurs que de celle du public, ont dû être sutmontées. L'auteur note un pourcentage d'échecs de 20 pour cent environ. Il souligne les raisons de certains échecs (moeurs particulières des familles espagnoles au cours des accouchements).

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J. RAVINA, P. DEVRAIGNE, S. TSOULADZE et J. COENCA (Paris). Les auteurs attirent l'attention sur l'importance pratique de la notion pavlovienne de type nerveux et sur l'utilité d'approprier les techniques de préparation aux modes d'apprentissage.

L'opposition établie entre la méthode d'éducation et méthode de suggestion tomberait d'elle-même si l'on considère que les sujets appartenant au type intellectuel sont plus sensibles à l'éducation consciente et les types artistes à la suggestion.

La diversité des types expliquerait la variété des résultats; la préparation faite sur un mode donné ne convient pas à tous.

M. HAY (Montluçon) Réfléxions d'un médecin praticien à propos de 1,000 accouchements par psycho-prophylaxie.

C'est encore au Dr. LAMAZE que P. MALCOVATI (Milan) rend hommage dans la communication qu'il fait au sujet de l'expérience qu'il poursuit à Milan depuis 1954. Plusieurs centres existent actuellement, dans lesquels plus de 2,000 accouchements ont été pratiqués. Quatre-Vingts pour cent des femmes préparées ont un comportement correct pendant l'accouchement (40 pour cent sans aucune douleur). L'auteur pense que le nombre des interventions (forceps, périné) est moindre que dans les accouchements antérieurs. Des recherches physiologiques (oxymétrie, tests de THORN, EEG, tests psychologiques) sont poursuivies parallèlement dans le but de contribuer à l'étude théorique et pratique de la méthode. Les problèms théoriques sont évoqués ainsi que les rapports entre la méthode pavlovienne et celle de READ. L'auteur reconnaît la priorité des idées de READ, mais se déclare un disciple de l'école de VELVOVSKI, dont la technique plus stricte, mieux fondée, représente pour lui l'avenir.

J. DE LEËWE (S'Gravenhage) retrace les progrès accomplis par l'accouchement sans douleur en Hollande. Cette méthode, introduite en 1953 par le Dr. LAMAZE, a acquis un développement égal à celui de la méthode de READ, appliquée depuis 1949.

I. VELVOVSKI (Kharkov) rappelle, dans un rapport qu'il envoie, que dès 1920 son maître PLATONOV préconisait l'emploi de méthodes hypno-suggestives dans l'analgésie obstétricale. Cette étape a été dépassée ensuite en U.R.S.S. grâce à l'application pratique du nervisme pavlovien.

Après un rappel d'embryologie comparée, l'auteur émet les hypothèses suivantes:

(1) La douleur de l'accouchement peut revêtir deux formes: la première serait dûe à une modification metrice à point de départ local, la seconde à une modification neurodynamique du système cortex cérébral—sous cortex.

(2) Dans la psycho-prophylaxie pavlovienne l'appel à la raison, les explications rationnelles jouent un rôle majeur. Les techniques hypnotiques sont abandonnées en raison de leur non-applicabilité en masse. L'effet principal porte sur la prise de conscience, sur la participation active et raisonnée de la gestante.

En conclusion de la séance, le Président de la Société Française de Médecine Psychosomatique, F. MOUTIER, souligne l'intérêt et le succès de cette journée et remarque l'importance des problèmes encore à explorer. Il propose, au nom de la Société, l'organisation d'un Congrès mondial consacré à ce sujet dans deux ou trois ans. Cette proposition est acceptée à l'unanimité.

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Compte rendu de la séance du 27 juin 1957

M. R. GIARD—Les facteurs somatiques allégués dans les déficiences génitales masculines.

Rôle partiel et souvent imaginaire des facteurs somatiques dans les déficiences sexuelles masculines. Importance de l'imprégnation hormonale prépubertaire et pubertaire, ainsi que d'une intégrité nerveuse. Modalités de ces insuffisance, portant surtout sur l'érection et ensuite sur l'éjaculation.

Classification de ces motifs organiques allégués:

Les insuffisances glandulaires et surtout testiculaires, parfois réelles, souvent purement fictives, entraînant des thérapeutiques aveugles, illogiques, dangereuses parfois aussi bien du point de vue endocrinien, et surtout au point de vue physhique en cas d'échec. Elles demandent à n'être faites que sous contrôle d'un endocrinologiste.

La masturbation: celles que nous voyons sont celles des sujets à la fois hyperémotifs et vigoureux sexuellement. Elles ne sont pas graves en elles mêmes; il faut rassurer le sujet sur leurs conséquences.

Les abus sexuels sont difficiles à prouver, la nature limitant automatiquement les possibilités de chaque homme. Mais des considérations temporaires d'ordre sentimental ou physique, ou des excès contemporains de petites périodes euphoriques chez les cyclothymiques peuvent être suivies d'une asthénie sexuelle qui est simplement liée aux manque d'incitations extérieures ou intérieures.

L'âge joue un rôle non seulement par lui-même, mais par la conscience qu'on en a, et les comparaisons instinctives auxquelles il entraîne.

Les maladies locales ne jouent un rôle qu'indirect par les craintes des conséquences personnelles ou familiales, ou leur caractère soi-disant honteux.

Les traitements locaux ne sont justifiés que si l'affection locale représente par elle-même un facteur inhibiteur. Les ultra sons sont une thérapeutique qui semble excellente.

Un bon nombre de ces malades relèvent de la psychiâtrie.

MM. L. CHERTOK et C. VEIL—De l'intérêt du diagnostic psychosomatique précoce en traumatologie.

Chez les accidentés du travail, un facteur psycho-pathologique plus ou moins important peut être fréquemment décelé. Pour éviter aux individus blessés des souffrances et à la collectivité une charge inutile, il importe d'en opérer le dépistage et le traitement. L'intervention psychiatrique peut amener à réviser certaines attitudes médicales et certains usages administratifs. Deux cas cliniques d'hystérie post-traumatique sont discutés; la guérison peut être très rapide si la psychothérapie est instituée assez précocement.

MM. A. MORALI-DANINOS, J. AUBRY et F. CERF—Psychosomatique et traumatisme (Travail de la clinique chirurgicale orthopédique et réparatrice de l'Hôpital Cochin. Prof. R. MERLE d'AUBIGNÉ).

La consultation psychosomatique de la Clinique Chirurgicale Orthopédique et Réparatrice de la Faculté a entrepris depuis 1951 la vérification systématique de certaines hypothèses étio-pathogéniques relatives aux polyaccidents, ainsi que l'étude psychosomatique des polyaccidentés.

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Le premier travail semble confirmer la conception de MARBE (1926) selon laquelle il existerait chez certains individus une tendance aux accidents se présentant comme un facteur personnel de prédisposition psychosomatique acquise. Ces vues ont été soutenues depuis par de nombreux auteurs et en particulier par l'un des auteurs de la présente communication depuis 1948.

Sur 100 sujets étudiés, on trouve 48 monoaccidentés, 30 biaccidentés et 22 polyaccidentés. L'hypothèse de la sensibilisation aux accidents se trouve semble-t-il confirmée puisqu'une portion réduite des sujets (22 pour cent) totalise presque la moitié des accidents (49 pour cent).

L'étude psychosomatique des sujets accidentés montre chez les polyaccidentés des éléments typiques de la personnalité qui ne se retrouvent ni chez les monoaccidentés, ni chez les sujets témoins, consultant pour des troubles psychosomatiques divers.

Les facteurs étio-pathogéniques relevés sont:

(1) Des facteurs prédisposants, dans lesquels les mécanismes de frustration et de culpabilité amènent la recherche inconsciente de situations accidentelles.

(2) Des facteurs déclenchants, consistant dans la résolution des conflits par l'action, c'est à dire dans l'extériorisation active, qui provoque l'acte dangereux, et enfin.

(3) Des mécanisms répétitifs, qui tiennent à ce que la déviation instinctive, qui a causé l'accident, tend à le répéter tant qu'elle n'est pas traitée.

Le rôle de la figure paternelle dans les conflits psychologiques menant à l'accident a été mis en évidence dans l'immense majorité des cas de polyaccidentés, à l'étude clinique et thérapeutique desquels un deuxième travail sera consacré.

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BOOK REVIEWS

A. G. OGILVIE and D. J. NEWELL: **Chronic Bronchitis in Newcastle-upon-Tyne.** E. and S. Livingstone, Ltd., Edinburgh and London, 1957. vii + 115 pp. 15s.

THIS is one of the most important studies yet to have appeared on the subject of chronic bronchitis. Its chief interest for readers of this journal however, is in its methodology, which is a model for any psychosomatic research involving medical, social and psychological techniques. The problems of the study of chronic bronchitis are common to many psychosomatic conditions, and particularly to bronchial asthma. It is a common disorder, difficult to define, and classify, causing much ill-health, with some relation to allergy, a vague genetic background, and socio-cultural implications.

The authors set out to make a survey of chronic bronchitis in Newcastle-upon-Tyne. A preliminary study was made of 100 bronchitics and 100 controls, or presumed non-bronchitics, and from the experience of this pilot study the broad lines of the survey were mapped out. The authors emphasize that every possible detail must be fully considered, and very complete preparation made before the field work is begun. It is hardly necessary to point out that for any such study of a common disorder it is essential that patients be selected from the general population at random, and seen in their homes. The factor of selection renders invalid, in general terms, many of the findings in studies based on hospital populations.

A systematic sample of 1 in 40 of the houses in Newcastle was taken, and within each of these houses, all the inhabitants were interviewed. This basic sample would probably contain "the correct proportion of people in 1, 2, 3, etc., person houses, an important fact in the design of any socio-medical survey in which the effects of heredity, nutrition or cross-infection may be relevant." And, one may add, of psychological factors. Preliminary interviews were carried out by health visitors, and from the households presumptive bronchitics and matched controls were collected. The bronchitics and the controls were then examined either at the hospital clinic, or in their homes, the doctor being originally ignorant of their status. Ninety-six per cent success was obtained for both groups—95 per cent for the controls and 98 per cent for the bronchitics. Observer error is taken into account in assessing the findings, and although sophisticated statistical techniques were not used much valuable information was gained from the survey.

Amongst some of the interesting findings is that there was an observer error of 17 per cent between doctor and health visitor; that twice as many men as women suffered from bronchitis. A personal history of allergy and a family history of bronchitis was more common in bronchitics than controls, and bronchial asthma was 8 times as common in the bronchitic group. Locality was important, as was social status.

It seems apparent that there is a relationship between bronchial asthma and chronic bronchitis, and many other studies have stressed this relationship. What is needed now is a socio-psychiatric study of chronic bronchitis and of bronchial asthma, using much the same methods as in this study. This small book is packed with information, has only 47 references in the bibilography and yet provides more "meat" than many a book of 500 pages. It must be read by all of us who have a special interest in chest illnesses.

DENIS LEIGH

J. R. EWALT, E. A. STRECKER and F. G. EBAUGH: **Practical Clinical Psychiatry,** Eighth Edition, The Blakiston Division, McGraw-Hill Book Co. Inc., New York, Toronto, London. xiv + 457 pp., $8.00.

THE first edition of this book was published in 1925, so that for over thirty years there has been a steady demand for it by readers, a mark of confidence which makes a reviewer's work a good deal easier. The book clearly contains what medical students and beginners in psychiatry need in the United States, and it is therefore of interest to see in this eighth edition how the current is flowing.

and how the book contrasts with British and European text books of a similar nature. One of the most refreshing aspects of American psychiatry is its basic eclecticism, for whatever may be said about the influence of psychoanalysis in the U.S.A., this book shows that a broader view prevails in some quarters, and has an appreciative audience. Naturally, there is a chapter on basic psychoanalytic concepts, but there are also chapters on the somatic, genetic and sociological aspects of mental disorder. The compression so inevitable in a textbook such as this is well handled, and although there are times when the material suffers from this, in general the treatment is adequate. For English readers the style is unsatisfactory, the formation of sentences often being slipshod, as though they have been dictated rather than written and re-written. The book has clearly fulfilled a need in its thirty years of life, and this last edition provides a good basis for the beginner to learn some of the facts, and fantasies, of present-day psychiatry.

DENIS LEIGH

K. SCHNEIDER: **Psychopathologie Clinique.** Translation from the German by J. P. LEGRAND. Editions Nauwelaerts, Louvain, 1957. vii + 188 pp., 110 Belg. Fr.

THIS is a French translation of the fourth German edition of KURT SCHNEIDER'S well-known book. Clear and concisely written, never too wordy or too abstruse, it stands alone as the best brief exposition of clinical psychopathology. Like so much of German psychiatry, it remains almost entirely at a phenomenological level. It is interesting to study the bibliography. There is no reference to FREUD or JUNG, or to any Anglo-American psychopathologists. The references are entirely German. One might be back in 1936—time seems to have stood still. But like all good clinicians KURT SCHNEIDER'S writing has a timeless quality about it—clinical medicine *is* timeless, and his book is as readable and relevant now, as it might have been 20 years ago, and will be 20 years hence. It has become a minor classic, and its translation into French follows translations into Spanish and Italian.

For those who require a brief, factual, didactic introduction to clinical psychopathology, there is no better book—it will not, however, satisfy those who have advanced beyond the early stages of psychiatric training or knowledge. The translation has been well and faithfully carried out.

DENIS LEIGH

R. A. SPITZ: **Die Entstehung der ersten Objektbeziehungen. Direkte Beobachtungen an Säuglingen während des ersten Lebensjahres.** Ernst Klett Verlag, Stuttgart. 110 pp.

RENÉ A. SPITZ is an Austrian psychoanalyst who has been working in America for many years. He has made interesting observations on the behaviour of infants in their first year of life, and has published several papers in English-language journals since 1945. A survey of his work appeared in the *Revue Française de Psychoanalyse*, 1954, under the title "Genèse des premières relations objectales." The present book is a translation of the French publication with alterations by the author.

SPITZ is an excellent clinical observer who has drawn attention to some interesting phenomena in infants. In particular, he has investigated the smiling responses which generally appear in the third month of life, though they have been noticed as early as the twenty-sixth day and may be delayed until the sixth month. He found that this smiling response is usually elicited by a nodding human face (whether familiar or strange, smiling or frowning); but a nodding mask, containing a crude indication of two eyes and a nose, can have the same effect. After the first six months of life the smiling response tends to occur only at the sight of a familiar face, and at the eighth month the infant begins to show anxiety when an unfamiliar face appears.

SPITZ has also emphasized the importance of good mother–child relations for the well-being and normal development of infants. He has tried to establish some causal connections between certain psychosomatic disturbances and anomalies of maternal care. The three-months' colic of infants is, for instance, considered to be causally related to maternal over-protection and the self-demand

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feeding system. Similar causal associations are asserted to exist between atopic dermatitis and maternal hostility exhibited as anxious solicitude; between rocking and psychopathic mothers whose attitudes alternate rapidly between caress and castigation; between coprophagia and cyclic maternal mood swings; between depression or marasmus and the withdrawal of maternal affection.

SPITZ is aware that these assertions can only be tentative. Yet his ambition to incorporate them into psychoanalytic theory, and perhaps to advance the theory, tempted him into pitfalls of rash and dogmatic statements which he could not substantiate and for which he has been criticized by S. R. PINNEAU (*Psychol. Bull.*, 1955, **52**, 429).

There can be no doubt that Spitz's clinical acumen is not supported by the balanced self-discipline that is required in the design and presentation of research. His enthusiasm and ambition often make him accept evidence which is far from reliable. In the beginning of the book he promises: "We have renounced the so-called clinical method which concerns itself with selected material"; and "the number of children observed by us has been sufficient to allow significant statistical conclusions." These promises have not been kept. SPITZ reports clinical observations on selected material; there are no adequate statistical conclusions; and his findings should therefore be regarded as no more than interesting suggestions which will have to be tested by more rigorous investigations.

F. K. TAYLOR

Ciba Foundation Colloquia on Ageing, Vol. 3: Methodology of the Study of Ageing. Edited by G. E. W. WOLSTENHOLME and CECILIA M. O'CONNOR, J. & A. Churchill Ltd., London, 1957. 202 pp. 32s. 6d.

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THIS is a verbatim report of the third Colloquium on ageing held under the auspices of the Ciba Foundation. Two earlier volumes had dealt with general aspects of ageing and with ageing in transient tissues. The subject of the present volume is dealt with in profound and extensive contributions from twelve eminent research workers of different nationalities, who are joined in frank discussions by several outstanding workers in the gerontological and allied fields.

It is impossible to summarize the papers read during the Colloquium, as they ranged from biological and genetic themes, through physiological and clinical subjects, to discussions on methods of studying age changes of human performance, emotion, and intelligence. There is no reference to any studies of psychosomatic ageing. Prof. F. VERZAR in his contribution on adaptation and ageing seems to approach psychosomatic areas when he points out that decrease in capacity for adaptation is the main characteristic of ageing. In reviewing methods used to delimit and understand this age-wise decrease, he points to the parallelism existing between diminished psychological adaptation, especially that due to declining memory function, and diminished powers of adaptation to variations of temperature and oxygen pressure, or to demands on heart, kidney or connective tissue fibres.

The book presents an account of the great difficulties confronting research workers in gerontology arising from the complexity of the subject, difficulties of sampling, and the special problems arising from the passage of time in longitudinal studies. The reader will find an up-to-date account of the ways in which the scientists taking part in the Colloquium are attempting to overcome these difficulties, but they may regret with the reviewer that some statements made by discussers have not been documented with references to the literature.

FELIX POST

P. DE VISSCHER: **L'antifeminisme des Intellectuels.** Editions E. Nauwelaerts, Louvain. $2.80.

THIS is an account of a questionnaire study of anti-feminist attitudes carried out in Belgium on five hundred well-educated men and women, resident in, and near, Brussels. The sampling appears to have been rather poor and dictated by necessity rather than choice; certainly the results cannot be taken as, and are not claimed to be, representative of Belgian opinion as a whole. The questionnaire, which appears to have been well-constructed on the basis of a detailed review of the literature—most

of which is, of course, American—consists of ten items which appear to form a scale of anti-feminism. Most of the book is given over to a very detailed and rather tedious discussion of the answers of different sub-groups to each of these questions; it is doubtful whether the small number of subjects involved, and the rough-and-ready method of sampling used, really justify such detailed consideration. In the final chapter, the intercorrelations of the items are given, as well as means and standard deviations of different groups for the total scale. As is so often the case in these studies, the findings appear somewhat commonplace; thus, men are a little more anti-feminist than women, a result which is surprising only because the differences are less marked than one might have expected. Other detailed findings relating to religion, and so forth, will not be of much interest to English readers, who are not likely to read this book unless they have a particular interest in the subject matter.

H. J. EYSENCK

W. SARGANT: **Battle for the Mind.** Heinemann, London, 1957. xxiv + 248 pp., (25 pp. of photographs), 25s.

BECAUSE of its semi-popular nature and topical interest, this book has deservedly received widespread attention in the press and on television. Although primarily addressed to the general public and hence to be judged accordingly, *Battle for the Mind* should provide inspiration to practitioners and research workers in a wide variety of professions. The book covers many subjects, ranging from a critical discussion of leucotomy, ECT and other forms of therapy to the techniques used in religious and political conversions, together with an interesting, if somewhat speculative, account by ROBERT GRAVES of brain washing in ancient times. There is a fascinating discussion of the methods used to obtain confessions by the police of both communist and certain western states. It is alarming to note that although the motivations are undoubtedly different the techniques used to obtain confessions are fundamentally similar.

Throughout this book, Dr. SARGANT adopts a systematic theoretical position; he eschews empiricism and bases his thesis upon the concepts of PAVLOV. To accomplish this formidable task he has, in the reviewer's opinion, considerably oversimplified the writings of PAVLOV and made it seem as if the works of PAVLOV were a model of precision and clarity from which unequivocal deductions may be made with a minimum of ambiguity.

The hostility that Dr. SARGANT's books has aroused in some literary and journalistic circles stems probably from prejudice and, in certain instances, certainly from ignorance. There is a repugnance in the western world to the acceptance of a mechanistic explanation of man's behaviour. It is acceptable to arrive at conclusions concerning behaviour in dogs, but—say the critics—it is morally indefensible to extend these techniques to man, who, unlike the animals, is presumed to have an immortal soul. Because of this prejudice PAVLOV's contribution has been greatly neglected, and, especially in the United States, a wave of psychoanalytic fervour has taken its place. It is unfortunate for the advancement of psychology, which claims to be a science, that vague, mystical and subjective speculations should take precedence over scientific method and validated predictions, however limited their range.

The other criticisms stem from a mixture of prejudice and lack of knowledge. It has been said that approaches such as that advocated by Dr. SARGANT are sterile and likely to be of little therapeutic value. In view of the acknowledged poor record of success of many of the more orthodox branches of therapy, this seems to be a particularly rash accusation; surely no therapist can conscientiously afford to condemn untried any reasonable avenue to the amelioration of his patient's condition. Another pattern of censure is to condemn Pavlovian psychology either because of PAVLOV's alleged inhumanity to his animals or because of its inhuman applications in communist countries. One might as well indict Rutherford for Hiroshima. The real need, however, is not for a partisan critique of the critics, but for further experimental and controlled study of diverse opinions of all schools, the only requirement being that their postulates are so expressed as to make them amenable to rigorous investigation.

With some justification it has been commented that Dr. SARGANT presents little evidence concerning conditionability in man. There is a wealth of data, mostly ignored by Dr. SARGANT (who suggests the need for research into these very topics) relating conditionability to personality, both

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normal and otherwise. For example, it has been found possible to simulate in normal subjects abnormal patterns of conditioned response behaviour by the use of various pharmacological agents. A remarkable similarity has been demonstrated between the conditionability of certain hysterics, psychopaths, and brain damaged subjects, on the one hand, and normal individuals under the influence of a depressant drug, on the other hand.

It is possible to account for these and other findings within the broad framework of Pavlovian psychology in much the same manner as Dr. SARGANT skilfully discusses the aetiology and treatment of wartime neuroses. For example, drugs may be prescribed on an empirical basis or their effects may be predicted in terms of some rationale and related to variables which are known to be relevant, such as personality. Thus the reviewer recently had occasion to suggest that a female introverted subject (hypothesized to be in a state of excessive cerebral excitation) be given a large dose of amphetamine sulphate. In accord with PAVLOV's principle of protective inhibition, which is presumed to supervene and protect the brain when stimulated beyond its capacity to tolerate the stresses imposed, the subject fairly soon fell asleep.

Far from being limited, as some pundits declare, the approach advocated by Dr. SARGANT offers increasing scope for an objective and rational investigation of the many problems of man's behaviour. Dr. SARGANT carefully avoids the common error of assuming that conditioning is the only technique by means of which the brain may be studied in the intact organism. It must be remembered that for PAVLOV, conditioned responses were merely one convenient method by means of which the general laws governing the mode of action of the central nervous system might be ascertained. It must also be remembered that abnormal psychology has progressed beyond PAVLOV into the wider field of learning theory in general. Only by utilising the discoveries of PAVLOV's successors (such as HULL) may Dr. SARGANT's important contribution be fully exploited.

CYRIL M. FRANKS

M. GROTJAHN, M.D., F.A.P.A.: **Beyond Laughter.** McGraw-Hill Book Company Inc., New York, Toronto, London, 1957. xvi + 285 pp., $6.00 or 45s.

THIS book is concerned with the dynamic psychology and unconscious meaning of laughter and of various related phenomena, such as jokes, the comic, humour, "kidding," the development of the sense of humour in the child, comedy, clowns, "strip-tease," Kilroy, Ferdinand the Bull, Mickey Mouse, and Alice in Wonderland. Although these subjects are treated consistently from a psychoanalytic point of view, it is an unusual and in some ways an unconventional book. Indeed the author says in his preface: "The unstated purpose of this book is almost a secret and it will not be mentioned hereafter. Reading this book should become an emotional analytic experience." In conformity with this, the book is written in a rather unsystematic, almost free-associative style, and the author recommends that it be read in the same spirit rather than from cover to cover. Undoubtedly this makes for easy reading; but as a conscientious reviewer I felt obliged to read it more conventionally. Doing this, one may be a little disconcerted by some of its features, e.g. the aphoristic style in which so many unsupported statements are made; and the difficulty of knowing when the author is restating the findings of FREUD and others or when he is putting forward his own views. An attempt is made to deal with the latter difficulty by means of notes at the end of each chapter, but only reference to the original articles cited would settle such doubts in the reader's mind. Clearly, one cannot have it both ways, but equally clearly the method the author has chosen makes his book more suitable as an introduction to the subject for the general reader rather than as a textbook.

Furthermore, readers of this Journal should perhaps be warned that the treatment is purely psychological, and that no reference is made to the somatic aspects of laughter, nor, for example, to the interesting connection between uncontrolled laughter and tears.

There is a very useful bibliography of 15 pages.

W. H. GILLESPIE

I. BELKNAP, Ph.D. **Human Problems of a State Mental Hospital.** McGraw-Hill, 1956. $5.50.

THIS book may well become a minor classic in the sociology of mental hospitals, a new territory of applied sociology which was pioneered by STANTON and SCHWARTZ in *The Mental Hospital* and by WILLIAM CAUDILL (the first social anthropologist to study a community of psychiatric patients from within, and to produce a report which could be published). Both of these prior studies had shown the role which social factors can play in exacerbating or relieving patients' symptoms, and stressed the importance of interpersonal relationships between different members of the medical and nursing staff. CAUDILL was able to go further, in his analysis of the informal sanctions which the group of patients imposed upon its members.

It could still be argued that the hospitals on which these first studies were based (Chestnut Lodge, a very expensive private hospital devoted to psychoanalytic treatment for psychoses, and a New England psychiatric clinic respectively) were special cases, far removed from the harsh realities of the average large American state hospital. This reproach cannot be levelled against Dr. BELKNAP's book. "Southern State Hospital," which was the subject of his three-year study, is grimly typical of American public mental health services. In the U.S.A. psychiatry is focused primarily on the lucrative treatment of private patients. It has been calculated that 85 per cent of American psychiatrists are engaged in treating 15 per cent of all the psychiatric patients in the country. Of the very large numbers of public patients, those who are eligible for treatment in Veterans' Administration are relatively fortunate because conditions in the State Hospitals, the repository of the underprivileged majority, are abysmal.

Southern State Hospital, for example, contains 58 per cent more patients than it was designed for, and many are accommodated under rudimentary conditions. Since its opening in 1861 the hospital's history has been one of inefficiency and neglect, revealed by a long succession of committees of inquiry, but never remedied. It is chronically understaffed. None of the junior medical staff, whose average length of stay is only eighteen months, are trained psychiatrists; they were found to be nominally responsible for the care of from 150 to 1,200 patients each, at different times during this study. The average stay of the medical superintendents has been 2½ years. There is a constant shortage of attendants, of whom 65 per cent leave before they have completed one year's service.

This is indeed a stark picture. By the medical and auxiliary staff, no less than by the patients, Southern State Hospital appears to have been regarded as a sort of penitentiary, to be escaped from as soon as possible.

When such conditions prevail, it is not surprising to read in the foreword that: "It is easy for state hospital superintendents to browse through the records of speeches of fifty or a hundred years ago and find quotations and recommendations appropriate to the problems which beset them today." Since the day of DOROTHEA DIX there has been no lack of exposures of the gross deficiencies of these institutions, but reforms have always been partial, temporary and ineffective. It is surely inadequate to ascribe it, as H. WARREN DUNHAM has done, to "some deepseated flaw in the administrative design or social organization of the mental hospital." The author of the present work comes nearer the mark when he writes: "What is done or not done in the hospital is determined by the way in which the institution has been conceived by the state and local governments and their citizens."

This phenomenon of the stubborn persistence of a condemned slum in public administration is itself of sociological interest; but it calls for the attention of a social historian, rather than that of an analyst of social structure. After the author has indicated these gross deficiencies in Southern State Hospital it seems curiously irrelevant that he should seek their causes, and their remedies, in "observations of the work and interpersonal relations" of a clearly demoralised hospital staff. The remarkable thing is that, even in so unpromising a setting, he has been able to do so much.

Dr. BELKNAP's study differs from previous investigations into the running of Southern State Hospital in that it does not stop short at demonstrating its deficiencies. The sociological analysis begins only after the exposure ends. Its achievement is to map very clearly the subterranean system of values and sanctions which enable this ricketty structure to keep going at all.

A sociological analysis can only take place, of course, where there is some element of continuity in an organization. The author found this in the "hard core" of attendants (18 per cent of the whole number) who stayed for many years. It is this nucleus of middle-aged men which effectively carries on the work of the institution. They view the transient medical staff with guarded hostility: "They don't know what goes on," they say, and the truth of this dictum is borne out by the present book. Dr. BELKNAP shows that the doctors' concept of the function of the hospital, of the classification of

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patients, and of their treatment is in large measure a fiction corresponding to their self-image as physicians, but not at all to the realities of life on the ward.

Many young psychiatrists, serving their apprenticeship on the wards of a large mental hospital, must often have had a suspicion that this was going on (just as the regimental medical officer guesses that his N.C.O.s largely decide what he will see and do). It is very difficult for the doctor, situated in his defined social role, to penetrate into this hidden world—though D. V. MARTIN, in his essay on "Institutionalisation" (*Lancet*, 1955, **2**, 1188) showed himself capable of doing so.

The present study shows that "what really goes on" in the wards is not haphazard, but obeys certain regularities. The key influences at work are the attendants' status system, their traditional concept of their role, and their opinions about mental illness. In terms of status, the attendants rank very low among hospital employees, but their self-esteem is buttressed by their position of authority over the patients; these in turn are ranked in a hierarchy as "co-operative," "not troublesome" and "unco-operative," in descending order. Attendants will summon patients by their Christian names, but do not expect to be similarly addressed in return. The attendants cling to their role as overseers: menial tasks must be done by patients. They regard the majority of long-stay patients as incurable, and as capable of sudden unpredictable outbursts of violence; in consequence, they believe that firm discipline should be enforced upon them. Attendants have great power over the patients, not only in their ability to give or withhold privileges and punishments (E.C.T. is widely used as a threat to enforce good behaviour), but also because the attendant effectively controls the patients' access to the doctor on his relatively infrequent visits.

Chapter 10 ("The Ward Control System in Action") brilliantly illuminates that side of mental hospital life to which the psychiatrist all too easily turns a blind eye. Although the picture it draws is grimmer, one hopes, than that which prevails in the great majority of British mental hospitals, it is still one which can be studied with advantage by everyone who finds himself in charge of long-stay patients.

Dr. BELKNAP has done an admirable job in analysing the functioning of a glaringly un-therapeutic community. As he sees it, the most serious of Southern State Hospital's many deficiencies lie in the lack of social therapy (since individual psychotherapy is clearly impractical with such large numbers). His remedy would be to create an entirely new role of social therapists, men and women with college training whose business it would be to practice group and occupational therapy with the patients: this should be linked with a very great increase in social work with the patients' families, with the aim of preventing the patients' being relegated to the hospital for good. These remedies are startling, and they reveal two prominent biases on the part of the author: (i) that social, rather than medical, treatment should take first place in the mental hospital, and (ii) that social science training is the best equipment for the psychiatric nurse of the future. These hypotheses are, to say the least of it, debatable: but the remedy he suggests does have the great merit of promising to break out from the *impasse* in which State Hospital practice has been stuck for so long. It is interesting to note its correspondence with the successful experiment carried out in a Canadian State Hospital by CUMMING, CLANCY and CUMMING, reported in their paper "Improving Patient Care Through Organizational Changes in the Mental Hospital" (*Amer. J. Psychiat.*, 1956, **19**, 249–61).

Dr. BELKNAP's book has thrown new light on a black spot in hospital psychiatry. It deserves to be widely read.

G. M. CARSTAIRS

Schizophrenia—Somatic Aspects. Edited by D. RICHTER. Pergamon Press, London, 1957. 181 pp., 40s.

THIS volume is the outcome of a discussion group held at the Ciba Foundation in London with the assistance of the Mental Health Research Fund. The present position of our knowledge of schizophrenia in the various fields of research in which they are engaged is reviewed by a number of leading English workers. The scope of the book is limited to somatic aspects and, whilst it makes no claim to be comprehensive, the subjects covered represent the most important fields of research enquiry. Included are chapters on physique, genetic and environmental factors, EEG findings, biochemistry, endocrine changes, pathological anatomy, neurophysiology and drug action.

In general a high standard of critical appraisal is maintained and excursion into speculation is only occasionally indulged. That so few positive findings emerge is a criticism of our present state of

knowledge rather than of the authors. Many workers have found hyper-, hypo-, and normal functioning for a variety of variables studied, but most have agreed to a greater tendency to individual variability in schizophrenics as a group. In the face of a mass of conflicting evidence the authors maintain a commendable caution in interpreting findings as cause rather than effect and due attention is paid to the limitations of methodology.

There are few who would now claim an exclusive somatic or exclusive psychogenic origin of schizophrenic but that there is some genetic or biological substrate is widely accepted. Yet schizophrenia is not a nosological entity and the schizophrenias are not a homogeneous group for experimental purposes. Research must in future attempt to define its experimental groups more clearly but perhaps only in the course of future research will groups, more significant than our present nosological categories, clearly emerge.

This book can be recommended as a reliable, up-to-date account of the state of our knowledge of the somatic aspects of schizophrenia.

BRIAN ACKNER

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J. Psychosomatic Res., 1958, Vol. 3, pp. 89–169. Pergamon Press Ltd., London

THE POST-CORONARY PATIENT

A MULTIDISCIPLINARY INVESTIGATION OF MIDDLE-AGED SWEDISH MALES*

O. FORSSMAN, M.D.† and B. LINDEGÅRD, M.D.‡

INTRODUCTION

THE increase in the incidence of myocardial infarction during recent years has been followed by intensified research into the causation of the disease. It is evident from multidisciplinary investigations embracing demographic, epidemiologic, anthropologic, genetic, and biochemical studies, that the causation of myocardial infarction is multifactorial and includes hereditary as well as environmental elements (for review see *inter alia* GERTLER and WHITE, 1954).

In the present study, postcoronary patients and age- and sex-matched controls were compared as regards occupational, genealogic, anthropo-somatologic, psychologic, and biochemical (including endocrinologic) variables. In addition, the relationship between the incidence of myocardial infarction and the occurrence of peptic ulcer was considered. Our results agreed on the whole with those of other investigations. Nevertheless, the study was extended to embrace different sub-groups of patients. Systematic analysis of such sub-groups showed it to be possible to distinguish at least two different categories of patients in the series under study.

The phenotype of one of these categories of postcoronary survivors (type I patients) was so conspicuous that it should be recognized readily even among persons in health.

We believe investigations of the kind reported in this paper to afford information making it possible to predict the danger of myocardial infarction in persons in health, thereby permitting the institution of prophylactic measures adapted to the individual case.

MATERIAL

Both patients and controls were collected from the residents of Malmö, a town in the South of Sweden with a population of some 200,000. As regards hospital services, Malmö is a closed unit. The town has only one hospital, which serves exclusively the residents of Malmö.

Examination of both patients and controls was carried out during the period October 1955 to March 1956. The patient group comprised all male survivors admitted to the hospital for myocardial infarction during the period 1948 to 1955 and whose age at the time of the examination did not exceed 55 years.

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The age distribution of the series is given in Table 1. The vast majority of patients (fifty-nine out of sixty-six) were between 46 and 55 years of age.

The examination was carried out within a minimum of 2 months and a maximum of 8 years of the initial onset of the acute myocardial infarction. In fourteen cases the examination was conducted within 6 months of the onset.

TABLE 1.—AGE DISTRIBUTION OF THE PATIENTS

Age group	No. of cases
31–35	1
36–40	1
41–45	5
46–50	27
51–55	32
Total	66

Since the variables to be studied vary with sex and age, the controls were selected in a manner which ruled out the influence of these factors.

The controls were chosen from the Municipal Register of Residents of Malmö in the following manner: the Register lists all persons resident within the town according to (a) date of birth, and (b) alphabetical order. The male next in order after the patient was chosen preliminarily, or, if he was unable or did not wish to cooperate (which applied in some ten instances), the next in order was selected. Each control was examined in the same way as the patients. If this examination elicited symptoms suggestive of cardiac disease, or signs of sclerosis of coronary vessels or the aorta (which also happened in some ten cases), the control was rejected and the next in the Register selected in his place. Persons with current somatic disease or systolic pressure at rest of more than 150 mm Hg and/or a diastolic reading exceeding 90 mm Hg were also rejected as controls.

All comparisons between patients and controls were made in matched pairs. In certain calculations *within* the control group, a further twenty-four controls were studied. These persons were selected from the Register on the principle described above as matching controls for a series of patients with general atherosclerosis in whom myocardial infarction was absent.

This latter series of patients, in which the age distribution was roughly the same as in the infarction patients, was examined separately but the results are not reported in the present paper.

Four of the sixty-six infarction patients lacked matched controls owing to the rejection of the controls for various reasons when the collection of the series had been completed.

The various sections of the examination were carried out at different times. Poor co-operation led in some ten instances to incomplete records. These persons are included in the analyses for which data were available.

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CLINICAL EXAMINATION

In the first session, the history was taken and a physical examination performed by one of us (FORSSMAN). History-taking was aimed primarily at eliciting data as to familial cardiovascular disease, prior or current somatic disease, and subjective cardiac symptoms, particularly cardiac decompensation (relative or absolute) or classic angina pectoris.

Physical examination included the heart, lungs, and abdomen; blood pressure at rest was recorded with Riva-Rocci's method. The heart and blood vessels were examined electrocardiographically and roentgenographically. The electrocardiographic recordings were made with a Triplex portable apparatus and three standard limb leads (leads I, II and III) as well as two chest leads (IV R and CR 7). The normal electrocardiogram was assessed according to GREWIN (1948).

All persons selected to serve as controls and the majority of patients underwent roentgenographic examination. The heart and thoracic aorta (frontal view) and the abdominal aorta were examined for the presence of intracardiac calcifications and calcific deposits in the aorta. The frontal views of the heart were taken with 0·028 to 0·035 sec, 95 kV, and 500 mA, and the abdominal aorta with 0·06 sec, 48 kV, and 125 mA.

At the initial examination, the subject was informed that psychologic and anthroposomatologic examinations would be made later by a different examiner (LINDEGÅRD). At this later examination, especial importance was attached to the fact that this examiner did not know whether the subject belonged to the infarction patients or the control group, or to the third group of patients (excluded in the present communication) in whom infarction was absent but who had symptoms of general atherosclerosis. The subjects were presented for examination at random, patients alternating with controls.

At the start of the interview, the subject was informed of the importance of not revealing the group to which he belonged. Only in occasional instances did he involuntarily let fall remarks disclosing his membership of one of the patient groups. As emerges in the following report on the psychologic rating, the investigation was aimed at eliciting certain habitual personality traits; the limitations placed upon the interview by witholding information as to the category of the subject can scarcely have affected the results.

The anthroposomatologic recordings were always made after the psychologic examination.

VARIABLES STUDIED

A. Occupational

The part-variables studied under this heading were leadership in the subject's occupation and the physical activity demanded by the occupation. The grading of these variables was based solely upon knowledge of the subject's occupation, not upon any detailed personal history.

The physical activity of the subject outside his occupation was, in fact, recorded, but these data were not included in the analysis. This is because any rating of such activity presents difficulty, since the non-occupational activity frequently varies appreciably in one and the same person from time to time. As might be expected, the participation in outdoor sports decreased from the ages of 25 to 30 years, although to widely varying degrees. During the years immediately preceding the onset of myocardial infarction, physical exercise was taken at varying intervals and only for short periods by many subjects. Consequently we do not believe the exclusion of non-occupational physical activity to constitute any *systematic* source of error of any magnitude.

Leadership was divided into three grades:

(1) *Leader*. This class included executives, managing directors, industrialists, heads of firms, dentists, etc.

(2) *Supervisor*. Departmental heads, foremen, elementary school-teachers, etc.

(3) *Subordinate*. Clerks, office workers, chauffeurs, industrial workers, unskilled labourers, etc.

Occupational physical activity was also graded into three classes:

(1) *Manual labour*. Unskilled labourers, builders, plumbers, welders, lorry drivers, etc.

(2) *Moderately heavy work*. Postmen, chauffeurs, factory workers, etc.

(3) *Sedentary work*. Business men, office workers, school-teachers, etc.

B. Genealogic

Both patients and controls were questioned as to the age at which their parents had died and the cause of death, by the examiner who collected the series (FORSSMAN). Special attention was given to cases in which either or both parents were reported to have died before the age of 71 of cardiovascular atherosclerotic disease (coronary disease, cerebral apoplexy). In instances in which no exact information was available, the Swedish Death Registration System was consulted for further data. The age limit was set at 71 entirely arbitrarily and is, of course, purely conventional.

C. Anthroposomatologic

Of the body tissues, it is mainly bone, muscle, and fat which underlie the somatologic differences between individuals. By recording the amount of these tissues present, and their distribution in the various regions of the body, it is possible objectively and metrically to define the individual somatologic character (LINDEGÅRD, 1953).

The definition of the individual characteristics may be more or less detailed depending upon the aim of the investigation, whether the body-build as a whole is being studied or only a certain portion.

For most purposes it suffices to record four main body variables (length, sturdiness, muscle and fat factors) according to the body-build diagram introduced by LINDEGÅRD (1953) (Fig. 1).

Length and sturdiness factors refer to the skeleton and are represented by the length and breadth of the long bones. The muscle factor refers to the cross-section area of certain groups of muscles of the extremities and is measured dynamometrically. The fat factor, finally, refers to the amount of body fat.

In the present study, the four main body variables are represented by the following dimensions:

Length factor: (a) right tibial length; (b) right radial length.

Sturdiness factor: (a) right femoral condylar breadth; (b) right bimalleolar width.

Muscle factor: (a) maximal hand-grip strength (right or left); (b) maximal shoulder-thrust strength; (c) maximal shoulder-pull strength.

Fat factor: The skinfold thickness at the following sites: (a) just below the right scapula; (b) on the right side of the chest at mamillary level; (c) on the abdomen, to the right of the umbilicus.

For details of the technique used in measuring, the reader is referred to an earlier report (LINDEGÅRD 1956). In order to allow of comparison with other studies, the above somatologic records were supplemented with data on body-length and body-weight.

As mentioned earlier, the examiner (LINDEGÅRD) was not informed whether the subject was patient or control so as to obviate any possible effect upon the results of the psychologic rating deriving from preconceived ideas. This exploration was always performed before the somatologic examination. In cases in which the dynamometric recording of the muscle factor might entail danger to the subject, the examiner was informed of this immediately before the test by the other member of the team who was usually present during the somatologic examination. As a result, determination of the muscle factor in some patients was not fully comparable with the rest of the series, in that maximal strength could not be measured. Dynamometric determination of muscularity is, then, less suited to a series of patients of the present composition. It is clear that this influences the results of examination, leading to a lower mean value for the muscle factor in patients than is in fact the case.

D. Psychologic

In the present study, although we used standardized questions in some measure, we tried to allow some freedom in history-taking in order to assess habitual personality dimensions. No use was made of testing procedures. The form of rating described above, of course, entails some degree of error dependent upon the subject's ability to grasp, weigh, and answer the questions. For this reason,

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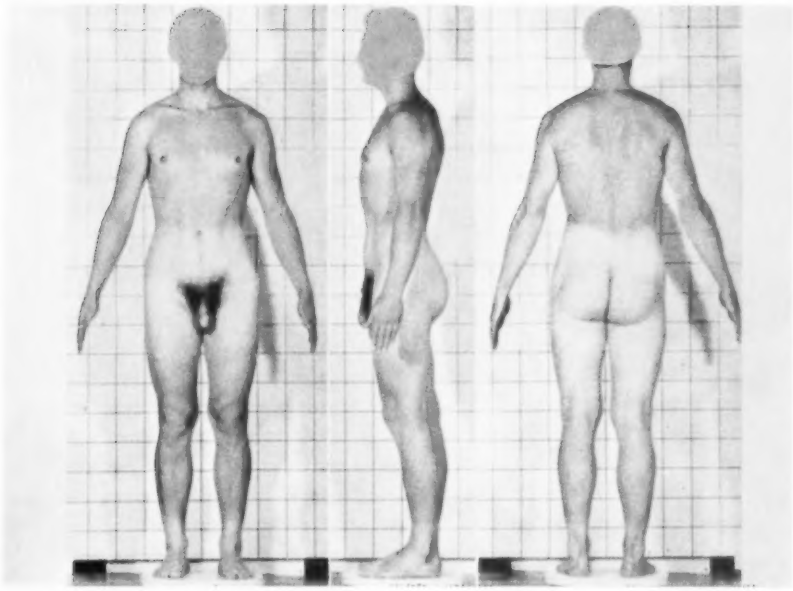

FIG. 1(a).—20-year-old male of average-sized length factor, but with large sturdiness-, muscle-, and fat factors. cf. Fig. 1(b).

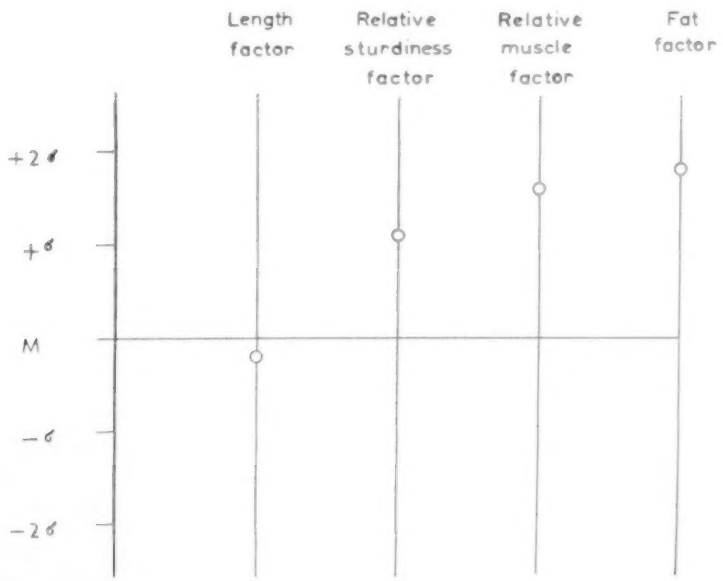

FIG. 1(b).—Body-build diagram for the individual in Fig. 1(a). All factors are given in standard deviation units. The factors were calculated as follows: Length factor from the radial and tibial lengths; sturdiness factor from the femoral and tibial condylar breadths as well as from the bimalleolar width; muscle factor from the physiologic cross-section of certain groups of muscles, taken to represent the maximal gross strength of the muscles; fat factor from body-weight, limb girth, or skinfold thickness.

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the rating of personality was supplemented with observation of the overt behaviour during the interview. NYMAN (1956) showed recently that behaviour diagnosis in young adults as a rule gives valuable information on constant individual personality dimensions. However, since the behaviour pattern may show considerable intra-individual variations from one short period to another, personality assessment was based mainly upon the personal history. In middle-aged persons, as were examined in the present study, there is also an appreciable danger that behaviour is affected by stereotype reactions deriving from environment, for instance from educational factors, and from the profession of the subject. Some personality dimensions were rated mainly on the history, others mainly on observation of the overt behaviour; this will be discussed in detail in the reports of the separate dimensions.

By withholding from the examiner information about the group to which the subject belonged, patient or control, we thought it would be possible to eliminate the systematic errors which are inherent in subjective rating when the interviewer has some knowledge of the subject and allows his preconceived ideas to influence his assessment. The only possible remaining cause of such halo effects could be via the communication of physical traits. As will be seen in the report on the results of the study, the differences in physical dimensions between patients and controls were not so marked as to allow of indirect rating of psychologic dimensions or differences therein.

(1) *Continuous psychologic variables.*

There is no certainty as to which psychologic variables should be regarded as continuous (i.e. present in every individual in a population but in varying degrees) and which should be considered as discontinuous (i.e. features either present or absent in individuals). To the purely discontinuous psychologic variables one should be able to assign such traits as derive from particular psycho-pathologic syndromes. On the other hand, other variables, such as intelligence, obviously vary continuously within the population. Finally, however, there are traits which cannot with certainty be assigned to one or other of the two groups. This is exemplified by one of the variables studied by us, 'explosivity'; as is discussed in detail in the following, this was recorded as a discontinuous variable. By 'explosivity' we meant threshold to stimuli leading to anger so low as to fall below the limit regarded as physiologic. Of course, one might just as well postulate explosivity as a continuous variable according to the higher or lower level of threshold to stimuli of the type mentioned above.

The reason for the methodologic dilemma mentioned above is the abstract character of the psychologic dimensions. Somatologic dimensions can be recorded quantitatively, their part-dimensions studied, and their interrelations analysed empirically. Most psychologic dimensions, on the other hand, can be metrically assessed neither according to their distribution within the population nor as regards their part-dimensions. However, on the basis of subjective ratings, such an analysis was carried out by NYMAN (1956). NYMAN adopted the hypothetical system of 'continuous' psychologic variables introduced by SJÖBRING (1913 and later). Assuming the variables to be the expression of a biologic continuity, it is an appreciable advantage from the methodologic standpoint also to record them as truly continuous and not as alternative variables. This facilitates the study of co-variations between the different variables, more so than if each variable is recorded as an alternative assessement. Another advantage of a system of continuous variables is that it allows the creation of a *personality profile*, a simultaneous representation of several variables in one and the same individual. A personality profile is, then, analogous with the body-build diagram mentioned above in the section on somatologic variables. This profile technique is now generally accepted in differential psychology, especially in intelligence research. Although the records are usually compiled with the help of test procedures and not by means of rating, the grading and frequency histograms in both methods are artificial constructions based on the hypothesis that the variable under study follows approximately the random distribution within the population.

In assessing the Sjöbring variables, each interviewer has his own hypothetical conception as to what constitutes the mean and the extreme values in the population. On this basis, each variable was rated according to a scale of seven grades as follows:

(1) Extreme deviation from the mean towards *lower* values.
(2) Marked deviation from the mean towards *lower* values.
(3) Possible deviation from the mean towards *lower* values.
(4) Mean value within the population.

(5) Possible deviation from the mean towards *higher* values.
(6) Marked deviation from the mean towards *higher* values.
(7) Extreme deviation from the mean towards *higher* values.

Grades 1 to 3 are termed sub-forms, grade 4 medio-form, and grades 5 to 7 super-forms of the variable under study.

Assuming the Sjöbring variables to follow the normal distribution, one grade in this scale corresponds approximately to one unit of standard deviation in standard scores.

In NYMAN's (1956) study on 320 Swedish Army men, the distribution incidence, variance, and mean values were analysed when the first eighty subjects had been rated. This analysis gave values against which the examiner could correct his conception of the hypothetical variable's statistic parameters the better to fit the binomial distribution. In the present investigation, the series was too small to allow of such a pilot study and subsequent scale correction. The examiner had to resort to his experience of other series.

The Sjöbring variables originate from certain psychopathologic syndromes. SJÖBRING believed, partly in conformance with JANET, KRAEPELIN and later KRETSCHMER, patients with a particular syndrome to have some personality qualities in common, at a time when they were free from their mental insufficiencies. Thus, in the neurasthenic, depressive, and hysteric syndromes, basic qualitites were supposed to correspond: these were later called subvalidity, sub-stability, and sub-solidity. These basic qualities allowed SJÖBRING to devise a theoretical system of reference, which in turn was used to conceive the opposite poles (later called super-validity, super-stability, and super-solidity) of the primary basic qualities. In addition, the oligophrenic syndrome, by way of analogy, allowed SJÖBRING to conceive the basic quality sub-capacity, and, later, its opposite pole super-capacity.

For more detailed study of SJÖBRING's hypothesis, the reader is referred to reviews by SJÖBRING (1913 and later), LINDBERG (1938), ESSEN-MÖLLER (1939), GOLDKUHL (1943), NYMAN (1956), and LINDEGÅRD and NYMAN (1956).

So the cluster of traits characteristic of each of the sub-forms is mainly observational and in some part it derives, as does entirely the cluster of super-forms, from the unitary theoretical interpretation introduced by SJÖBRING. Thus, the results obtained with SJÖBRING's procedure differ fairly widely from those arrived at with methods in which clusters are built up from observed traits exclusively. This is exemplified below.

The variable cerebrotonia (SHELDON, 1942) embraces a part-variable—a tendency to abstract as opposed to concrete thought. In SJÖBRING's personality profile we find the tendency to abstract thought in two of the four variables: 'capacity' and 'stability' (see below). In this instance, SJÖBRING believes it possible to distinguish between different kinds of disposition to abstract thought, while SHELDON regards the trait as more symptomatic and assigns it to a single cluster.

On the other hand, in his system of reference SJÖBRING justifies the clumping into a common cluster of part-variables which, from a symptomatic standpoint, appear to be little related. Thus, the variable 'solidity' (see below) embraces a tendency to a higher or lower degree of subjectivity together with a higher or lower rate of association among other qualities.

In the present paper, discussion of SJÖBRING's personality profile is limited to a brief description of the extreme variants at each pole of all four variables (Table 2). In this connexion it should be stressed that, for reasons given above, the description of each extreme variant in the table should be regarded as an entity and the various adjectives should be taken as standing for one common basic tendency. No single adjective or group of adjectives should be distinguished as a part-dimension of the variable as a whole.

Capacity denotes the general level of intelligence of the subject. Here, the examiner based his estimation chiefly upon the personal history, such as reports on the subject's success at elementary school. Special attention was paid to his progress in mathematics. Further, the interviewer studied the subject's general maturity in discussing the factors underlying his choice of profession, etc. Here, then, the subject's behaviour during the interview was taken into account when rating Capacity. In instances in which capacity was rated low it is not clear whether the low intelligence level was the product of a primarily low degree of intellectual capacity or of an intellectual reduction brought about by the cerebral atherosclerosis or some other cause. This implies that the assessment was not independent of the subject's current level of intelligence, in that his ability to grasp and answer abstract questions, as also his weighing of problems affecting his situation, influenced the final rating.

Validity has, according to SJÖBRING, symptomatic traits in common with other personality

variables as defined by different authors (see NYMAN, 1956). Validity is, then, more or less covered by the following dimensions: HEYMANS' (1906 and later) activity, BURT's (1954) asthenia-sthenia, SHELDON's (1942) somatotonia and CATTELL's (1950) factors *E* and *J* (dominance versus submissiveness and neurasthenia versus vigorous, obsessional determination).

Validity was assessed mainly on the basis of the personal history. The questions were directed to estimating the degree of confidence or uncertainty in making decisions, or of behaviour in various

TABLE 2

	Super-types	Sub-types
Capacity	Talented, sensitive, responsive, perceptive, subtle	Obtuse, stolid, impercipient, dull, primitive
Validity	Alert, enterprising, effective, self-reliant, presumptuous, impetuous, tenacious	Quiet, cautious, unobtrusive, scrupulous, considerate, conscientious
Stability	Cool, abstract, and discursive mind; sophisticated	Warm, heavy, concrete, naive, clumsy, industrious, social, tending towards personal relationships
Solidity	Steady, consistent, dependable, circumspect, dry, objective	Agile, subjective, dramatic, pleasing, narrow, egocentric, impulsive

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social connexions. The interviewer tried at the same time to judge with current psychiatric methods whether or not a lack of confidence or self-reliance was the product of something other than subvalidity, such as neurasthenia deriving from atherosclerosis or an induced 'neuroticism'.

In assessing validity, great importance was placed on the results of discussion on the working conditions of the subject. By asking the subject's views on the monotony or interest in his work, on his preference for his subordinate or administrative status, and on the degree of his material and personal responsibility, the interviewer sought information upon which to base his rating of validity.

Stability in SJÖBRING's personality profile has traits in common with SHELDON's (1942) viscerotonia, CATTELL's (1950) factor *A* (cyclothymia versus schizothymia), and EYSENCK's (1947) extroversion-introversion. It should be pointed out that the variants of low Stability, or substable persons, appear to have many traits corresponding to what BLEULER (1922) described as syntonic personality, and KRETSCHMER (1921) as cyclothymia.

In the present study, the degree of stability was assessed by evaluating both the history and the overt behaviour of the subject during the interview. As was pointed out above in discussing general aspects of psychologic rating, estimation based on historical data contains a number of sources of error deriving from the subject's ability to grasp and answer the questions asked. In our experience, the danger of interference from such sources is particularly great when probing stability. We have not infrequently observed a striking discrepancy between the subject's report and his behaviour during the interview. Thus, the rating of stability demands of the subject a great degree of introspective participation in analysing his own attitude. This applies particularly to such questions as are directed to exploring his tendency to emotional engagement or neutrality. Even when the subject's intellectual capacity is such as to permit participation in such analysis, another source of error creeps in. This is related to his desire to present his own attitude as advantageously as possible from the social and conventional standpoints. This source of error, of course, applies equally to the other variables studied, but its effect is in our experience especially marked when assessing stability.

In order to eliminate as far as possible these sources of error, it appears to us reasonable to let the overt behaviour during the interview be more or less decisive when rating stability, and in any case to let it supplement the history. We thus gave special attention to the subject's general behaviour, his emotional heaviness, heartiness and accessibility in conversation.

LINDEGÅRD and NYMAN (1956) have shown that stability rating based exclusively upon such diagnosis of behaviour is capable of detecting elements characteristic of and constant in the individual. In their study on 20 year old Swedish Army men the degree of stability rated in this way was found to be related to the steroid fractions excreted in the 24-hour urinary volume, among other things. Later, LINDEGÅRD (1957) found comparable psycho-morphologic and psycho-physiologic correlations in a series of similar composition (Fig. 2).

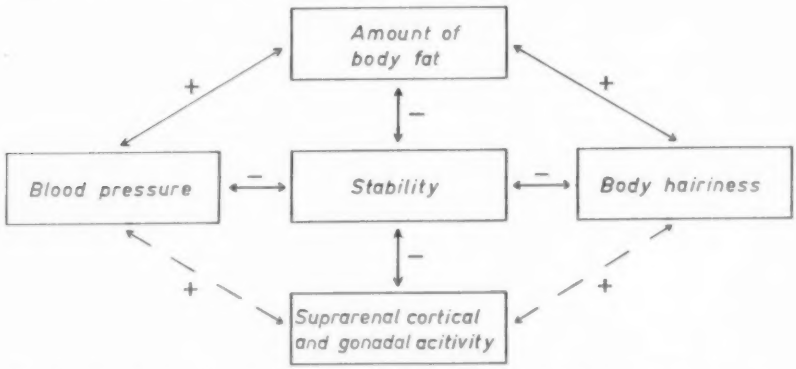

FIG. 2.—Correlations determined empirically are marked by unbroken lines; broken lines represent such co-variations as might be expected in the light of patho-physiologic experience. Positive and negative correlations are expressed by plus and minus signs.

Since in the present investigation stability was in part assessed by observation of the subject's behaviour during the interview, the possibility cannot be ruled out that a low rating may not fully reflect the earlier behaviour of the subject. It may derive in part from a mental change of later age (resulting from atherosclerosis or some other cause) which gave rise to some trend taken symptomatically to denote sub-stability.

Solidity appears to be closely allied to a variation between the two extremes 'dominance of primary function' and 'dominance of secondary function' as described by HEYMANS (1906, quoted from NYMAN, 1956). Further, a low degree of solidity appears to have features in common with the personality trait of '*Ungebundenheit*' described by V. BAEYER (1935) in *Schwindler und Lügner*. The extreme values for solidity variation correspond to the symptomatic personality characterization given for persons reacting extremely well or extremely badly to medication with placebo (LASAGNA *et al.*, 1954). Compared with the other variables in SJÖBRING's personality profile, Solidity appears to have been given less attention by other authors.

Solidity, like stability, was rated by evaluating historical data against the overt behaviour during the interview. In assessing solidity, the aim was to get the subject to report his concrete behaviour in certain stereotype social situations. A lesser degree of introspective participation was called for than that needed in rating stability.

Compared with observations made in similar studies in younger persons, the present investigation has given us the impression that the rating of solidity based on observation of overt behaviour during the interview deviates from that based on historical data more markedly in older than in young persons. If this impression could be verified, it might be accounted for by a modification of the personality profile with advancing age by the superimposition of a more stereotype pattern suited to the subject's age, social position and profession. This may, of course, apply in principle to all the variables studied but is perhaps particularly true of solidity owing to the fact that SJÖBRING read mental flexibility into this variable. An individual with low solidity, a sub-solid person, has according to SJÖBRING among other things a tendency to immature, impulsive reactions. Since he at the same time possesses a high degree of flexibility, he may assume an attitude of considered thoughtfulness and general maturity if *he himself* thinks such an attitude to be demanded by his social position or profession. In view of the general impressions mentioned above, the solidity rating in the present investigation was based solely on historical data.

As mentioned earlier, estimation of any component of SJÖBRING's personality profile is not founded

on isolated traits but mainly on an interpretation of the interrelations between different features observed. The method, then, is in principle fully comparable with differential psychologic systems (such as KRETSCHMER's, JUNG's, HEYMANS' and others) and with common somatic diagnosing as well. On the other hand, it differs appreciably from the methods usually applied in differential psychologic research.

As emerges in the foregoing, in practice the method frequently involves grouping into clusters behaviour qualities which are not closely semantically or symptomatically allied, while qualities symptomatically more nearly related may be dispersed. From the methodologic standpoint this must obviously give rise to doubts. On the other hand, it must be admitted that interpretation and systematization of observable mental symptoms may be capable of giving consistent results unobtainable with *a posteriori* systematization such as factor analysis. The chief methodologic objection to SJÖBRING's variables is the unverifiable degree to which the imagination and interpretation of the examiner is allowed free play as regards the interrelations between observed isolated mental phenomena.

The hypotheses put forward by SJÖBRING can be neither verified nor refuted empirically (NYMAN, 1956). On the other hand, the results of assessment can be evaluated *empirically* from the methodologic standpoint. Studies by LINDEGÅRD and NYMAN (1956) and LINDEGÅRD (1957) have led to attempts to analyse empirically the results obtained by various authors. The technique is founded upon verifying certain *multiple* correlations in each investigator's series between the variable rated and morpho-physiologic dimensions recorded metrically. Positive results have as yet been obtained only as regards Stability (LINDEGÅRD, 1957).

In the ideal estimation of SJÖBRING's personality profile, one must expect to find no demonstrable correlations between the different variables.* The lower the degree of such gestaltic interpretation to which the observed symptoms are subjected, the more symptomatically descriptive the estimation of the variable, and the more often one and the same symptom will be assigned to two or more variables and thus give rise to a correlation.

In studies of the relation between estimations of the Sjöbring variables published by ESSEN-MÖLLER (1956) and NYMAN (1956), the correlations between certain variables are stated to be significant, although they were different in the two studies. In the present investigation, there is a positive correlation between Capacity and Stability ($r = 0{\cdot}3$) and a negative correlation between Validity and Solidity ($r = -0{\cdot}2$), only the latter value agrees with that estimated by NYMAN (1956).

In the interpretation of the interrelation between isolated symptoms it is of importance that the four components of the personality profile are rated simultaneously. This counteracts the tendency to estimate the Sjöbring variables purely symptomatically, which might in turn in extreme instances lead to illusory incompatibilities.

(2) *Discontinuous psychologic variables*

During the earliest stages of the investigation, only three discontinuous variables were recorded: the presence of 'neurotic traits', 'impairment' and 'explosivity'. After a short period it was found desirable to increase the number since explosivity was found to be relatively common and, by using additional variables, it was hoped to differentiate the various sub-forms of explosivity.

The division of psychologic variables into two groups, continuous and discontinuous, was done chiefly for conventional reasons. Only those variables included in Sjöbring's personality profile were assigned to the group of continuous variables. It should be noted that other authors have classed some of our 'discontinuous' variables as continuous. MILES *et al.* (1954), for example, studied a continuous aggressivity variable which in some measure corresponds to our discontinuous variable explosivity. The presence of neurotic traits was recorded in the present investigation as a discontinuous variable.

Neurotic traits. The presence of neurotic traits was diagnosed chiefly by observation of the subject during the interview. The basis of estimation was similar to that used by NYMAN (1956) in his study of Army men. In accordance with NYMAN, expressive behaviour was termed neurotic if interpreted by the rater as expressing uneasiness, sadness, tension, affectedness, in a fashion that is

* A correlation significantly different from zero between two of the constituent variables implies an imperfect interpretation of the observable mental symptoms in the subject. The degree of such a correlation may, consequently, also be said to be a measure of the interviewer's *personal error* in this respect. On the other hand, the absence of interrelation between the variables is not equivalent to an ideal estimation.

obviously observable even when occasional diverting stimuli change the habitual attitude (cf. EYSENCK, 1947, dysthymic neurosis).

Impairment. This quality was assessed exclusively by observing the subject during the interview. 'Impairment' was recorded as a marked discrepancy between the subject's relatively low capacity for abstract thought and relatively high degree of general intellectual gradation in his behaviour—including such factors as his vocabulary.

Symptomatically, impairment recorded by us appears to have traits in common with those seen in patients with slight organic deterioration. Most often, persons with impairment have a tendency to what GOLDSTEIN *et al.* (1941) described as the 'concrete behaviour' of persons suffering from cerebral lesions (cf. G. UDDENBERG, 1955; NYMAN, 1956; DENCKER and LÖFVING, 1958).

Impairment should not be confused with a low degree of capacity. Despite the presence of low intellectual power as estimated by behaviour diagnosis, one finds in the history of persons with impairment a report of earlier comparatively high intellectual powers.

Periodic depressions. The presence of this quality was estimated from the history on the following symptoms: recurrent, short-lived depressions (of 2 to 5 days' duration) together with a general feeling of mental unease and consciousness of a decrease in mental and bodily powers. A prerequisite for the acceptance of 'periodic depressions' was that neither the beginning nor end of the period should be closely related with changes in the subject's environment.

Explosivity. When exploring for explosivity, the interview was directed to demonstrating a low threshold of irritation to stimuli producing abrupt, involuntary anger. The presence of explosivity was rated according to the interview.

This aspect of explosivity has traits in common with the quality termed irritability by MAYER-GROSS *et al.* (1954). It would appear, however, that the irritability as defined by these authors is a less specific trait than that covered by our term explosivity. According to MAYER-GROSS *et al.*, the outbreak of anger in irritable persons leaves behind either euphoria or a sense of depression. The quality recorded by us, explosivity, embraces as an obligatory symptom a marked feeling of embarrassment after the outbreak. This implies that the subject himself feels that the anger is an excessive reaction in relation to the magnitude or intensity of the precipitating stimulus. Further, we made it a condition that the outbreak should follow the stimulus without any noteworthy period of latency and that the emotional reaction should be of short duration, in other words it should in truth resemble an explosion. The chief traits in the aggressive behaviour defined can be summed up as follows: low threshold of irritation, acute arousal of emotion, short duration, and subsequent embarrassment.

In the study, no attention was paid to the nature of the provoking stimuli. The formal quality of the emotion was studied by a concurrent recording in the greater number of subjects of a number of variables taken by certain authors (STRÖMGREN, 1936; MINKOWSKA, 1937) to be a delimited syndrome together with explosivity. This syndrome has frequently been described as connected with epilepsy and its traits have, consequently, been termed epileptoid. STRÖMGREN introduced the expression ixothymia; SJÖBRING extended the term and denominated it ixophrenia.

In addition to explosivity, the following traits have been distinguished in this syndrome: oversensitivity to injustice, inability to forget suffered injustice, cumulation of emotional tension, intensity, and viscosity.

In our analysis, we did not treat these personality traits collectively as a syndrome, but recorded each symptom separately concurrently with the estimation of explosivity. The following comments refer to the principles underlying this rating.

Oversensitivity to injustice. The subject was asked about his emotional reaction to experienced, concretely defined social situations which implied that he had been unjustly overlooked, wrongly accused, or otherwise treated with injustice, as well as about his reaction when witnessing another person in a similar situation. From the report on the subject's habitual emotional attitude to these and comparable situations, we attempted to decide whether or not the threshold of irritation to these stimuli was lowered. This variable, then, was rated exclusively from the personal history.

This exploration was closely followed by examination as to the subject's ability to forget and free himself from aggressivity resulting from oversensitivity to injustice. In those to whom 'inability to forget suffered injustice' was ascribed, it was noted in the history that wholly insignificant trifles were borne for a long time, in some instances for several years.

When compiling data referring particularly to the two variables mentioned above, it was noted

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whether the subject's emotions were aroused at the memory of situations of the relevant type. The subject frequently linked the more stereotype situations evoked by the examiner with concrete situations which he himself had experienced. Reactivation of aggressivity in this manner was noted as 'cumulation of emotional tension'. Emotional arousal was relatively uncommon compared with the traits mentioned above. It was usually associated with a marked habitual 'intensity' in mode of expression even when emotionally neutral subjects were discussed.

Symptomatically, this intensity—as an isolated phenomenon—is scarcely distinguishable from the intensity or vitality which is part of the behaviour pattern of a person with high validity as rated by SJÖBRING, or with marked somatotonia as defined by SHELDON (SJÖBRING, 1955, personal communication). Since intensity was regarded as a part-phenomenon of the ixophrenic or epiliptoid character, it was recorded separately in the present study. However, positive findings were noted only in cases in which the examiner considered the degree of intensity to be strikingly high in relation to the rated degree of validity (see above). As mentioned earlier, validity was rated chiefly from the personal history. 'Intensity', however, as also 'cumulation of emotional tension' was estimated exclusively from the overt behaviour of the subject.

'Viscosity' was also assessed from overt behaviour. To denote a person as viscous, there had to be marked sluggishness in his general behaviour pattern. In advanced instances, one found a slow, hesitant manner of speech, pronounced difficulty in passing on from current thoughts and emotions, and a corresponding difficulty in grasping a new line of thought. The general movements and gestures of these persons including the play of features also show a sluggish pattern of reaction.

There is a certain amount of overlapping between the behaviour pattern of the viscous person and that demonstrated by an individual with high degree of solidity. To avoid confusion between the two variables, the traits described above were accepted as viscosity only in those instances in which they contrasted strikingly with the level of the subject's solidity.

E. Biochemical

Serum cholesterol level was determined according to ABELL *et al.* (1952). The sample was always taken in the morning before the subject had eaten. Repeated sampling on different days was as a rule not possible.

Urinary hormonal excretion. The following steroids were determined in samples of the urine (mg/24 hr volume).

Neutral 17-*ketosteroids* (17-KS). Extraction according to JENSEN and TÖTTERMAN (1952) and colour reaction with the ZIMMERMAN (1935) reaction as modified by CALLOW *et al.* (1938). Certain subgroups of the neutral 17-ketosteroids were also studied:

(a) 'Non-acid fraction' (NF) according to a method described by JENSEN and TÖTTERMAN (1952). These fractions are composed of steroids liberated at the preliminary hydrolysis of the urine by heating without the admixture of acid.

(b) 'Acid fraction' (AF), also determined according to JENSEN and TÖTTERMAN (1952). These fractions consist of 17-ketosteroids liberated at the subsequent hydrolysis of the urine by heating and acidification.

(c) Dehydro*iso*androsterone (DHIA) according to JENSEN and TÖTTERMAN (1952).

17-*Ketosteroidogenic steroids* were determined according to NORYMBERSKI (1952) and include such corticosteroids as give neutral 17-ketosteroids after oxidation with sodium bismuthate.

Tetrazoleum-reducing corticosteroids (TZR) were determined according to JENSEN (1958) during the latter part of the investigation in thirty-seven patients and thirty-seven controls when this method was introduced at the hormone laboratory.

Certain steroid substances are found in several of the fractions mentioned above. Even though there is no close chemical relationship between different steroid fractions, there may exist a biosynthetic connexion (GENELL, JENSEN and LINDEGÅRD, 1956). In order to obtain a comprehensive picture of steroid metabolism, it is desirable to study such fractions as are excreted in mutually independent quantities. On the other hand, the examination should for practical reasons be simplified by excluding fractions closely associated with others. Consequently, the biosynthetic relationship between the various steroid fractions was studied by calculation of the inter-individual correlation coefficient between these fractions (Table 3).

The steroid fraction termed AF showed a very close association with the 17-KS fraction; for this reason, the AF fraction was excluded from further statistical analysis.

TABLE 3.—CORRELATION BETWEEN VARIOUS STEROID FRACTIONS

	17-KS 17-KGS	17-KS DHIA	17-KS NF	17-KS AF	17-KS TZR
Patients	+0·47	+0·53	+0·72	+0·98	+0·16
Controls	+0·55	+0·64	+0·72	+0·95	+0·23

GENELL *et al.* (1956) have shown that some of the steroid fractions are normally distributed in the population and some skew-distributed. The statistical treatment of the data was, therefore, preceded by an analysis of the frequency distribution of the various steroid fractions. The fractions with skewed distributions, i.e. NF, DHIA, and TZR, were transformed to a scale with a normal distribution by the method of cumulative distribution before the calculation of correlation coefficients.

STATISTICAL METHODS

In comparative studies of the continuous variables in the different groups, three methods were used:

(1) Comparison of the arithmetic means for variables of normal distribution. The difference was subjected to the *t*-test (FISHER, 1941, and others). The same method was also applied to variables of skewed distribution, when these had been transformed to normal distribution by the cumulative distribution method.

(2) Intra-pair comparison between patients and their matched controls. The difference between the patient and control was determined only alternatively: 'larger than', 'equal to', and 'less than'. An appreciable preponderance of 'larger than' over 'less than' suggests a true difference. The significance was checked according to the χ^2-test, performed with the aid of a table of significance limits (DIXON and MASSEY, 1951, and others).

(3) Comparison of the number of high, medium, and low variants in various groups; significance was checked with the χ^2-test. When the group numbered less than five members, the exact treatment of 2×2 tables was used (FISHER, 1941, and others).

Since, according to the null-hypothesis, no difference was to be expected between the patient and the control groups, the boundary values for high and low variants were calculated collectively for the entire series of patients and controls. The limits of a half-unit of standard deviation above and below the arithmetic mean were taken to distinguish the high and low from the medium variants. When the variable was skewly distributed, the corresponding values were calculated with the cumulative distribution method.

If the probability due to chance was less than 1·0 per cent ($p < 0{\cdot}01$) it was termed highly significant; if it exceeded 1·0 per cent but was less than 5·0 per cent ($0{\cdot}01 < p < 0{\cdot}05$) it was termed significant.

For a correlation reported between two variables but without any specification as to the degree of significance p may be taken to be greater than 0·05.

COMPARATIVE STUDIES BETWEEN INFARCTION PATIENTS AND CONTROLS

This section of the investigation was aimed at demonstrating differences, if any, between post-coronary patients and normal controls as regards the variables discussed in the foregoing.

Only patients for whom there were age- and sex-matched controls were included in this study (sixty-two out of sixty-six). The somatologic and psychologic examination were not performed in seven cases (three patients and four controls); no serum-cholesterol determination was made in eleven cases; and no urine collected for determination of steroid excretion in two cases. Only matched pairs of patient-control were included in the study of these variables.

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Occupational variables. The part-variables studied were occupational leadership and occupational physical activity.

As regards leadership, there was a significant difference between the patient and control groups, the tendency being towards a higher degree of leadership in the patient group. The ratio of leaders to non-leaders in the patient group was 14 : 48, and in the control group 7 : 55. The results agree with the view held generally and verified in clinical series (HEDLEY, 1939; DUBLIN *et al.*, 1948; RYLE *et al.*, 1949; SMITH *et al.*, 1951; GERTLER *et al.*, 1954; *Oslo Investigation*, 1956; and others), that coronary thrombosis is common among business men, doctors, lawyers, officers, and the like.

The degree of physical activity demanded by the occupation is regarded generally as being somewhat lower in the category of person with a high degree of occupational leadership, and vice versa. Nevertheless, no difference was demonstrable between patients and controls as regards the degree of occupational physical activity.

Our results do not confirm the observations of MORRIS *et al.* (1953) on part of the population of a large city. The principles applied in their investigation differed from ours in that their series was drawn from definite occupations, the employees of the London Transport Executive and the General Post Office in London. Groups of persons with varying occupational physical activity were compared in each of the two concerns. In the section of their more comprehensive study dealing with male post-coronary patients below the age of 55 surviving the acute onset, there was a predominance of persons with heavy physical work. However, it is not stated whether this difference was significant.

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Genealogic variables. Data on the age and cause of death of either or both parents were lacking in four cases among the patients, as against one case in the control group. In seventeen cases in the patient group, and eight in the control group, one or both parents' death had been caused by atherosclerotic cardiovascular disease before the age of 71. In three of these seventeen cases among the patients, but in none of the eight control cases, *both* parents had died before the age of 71 of a disease of this type; in the remaining cases, the death of only one of the parents was attributable to this cause. The high parental mortality in the patient group was significantly higher than in the control group.

Somatologic variables. No significant difference was demonstrable as regards the somatologic variables by comparative study either of the mean values for the two groups or of matched pairs of patient and control. However, a certain tendency to deviation did emerge on comparison of the matched pairs, as regards the fat factor and body-length. This tendency was suggestive of a higher fat factor and lower body-length in the patients as compared with their controls. On calculating the frequency of extreme variants in the two series, one of these tendencies was verified, in that the patient group showed a higher incidence of obese persons; this tendency was highly significant. The ratio of obese to non-obese persons was 28 : 27 among the patients, and 15 : 40 among the controls: the fat factor was measured by the skinfold thickness just below the scapula.

Psychologic variables. Since the patient group showed a high incidence of occupational leadership, the average personality pattern in this and the control group was expected to differ. However, no difference attributable to this factor was demonstrable.

Of the discontinuous psychologic variables, one trait—the disposition to 'periodic

depressions'—showed a highly significant difference between the two groups. This dimension was not explored separately in every subject but was rated concurrently with the other alternative variables during the course of the investigation (see above 'Variables studied'). This led to a somewhat uneven distribution of patients and controls, more controls than patients being explored as to this quality. Of the fifty-three controls rated, only three were found to suffer from periodic depressions, whereas the corresponding figures for the patients were 12 of 38.

As is shown in the following, the tendency to periodic depression in post-coronary patients was usually combined with a low degree of stability. No such association was found in the control group. This group difference implies that the examiner, who did not know to which category the subject belonged at the time of the interview, rated the tendency to periodic depression independently of the degree of stability.

None of the continuous personality dimensions showed any significant difference, or tendency to any such difference, in the two groups. This applied whether the estimation was based upon the arithmetic mean of the group, comparative study of matched pairs, or analysis of the incidence of extreme variants in the two groups.

Biochemical variables

(a) *Serum cholesterol level.* Comparative studies in patient–control pairs, as well as the number of persons with hypercholesterolaemia (more than 300 mg cholesterol per 100 ml) in the two groups, showed there to be significant differences between the two groups. The patients usually showed a higher cholesterol level than their matched controls (in thirty-four of fifty-one pairs). Hypercholesterolaemia was present in seventeen of the fifty-one patients, but in only six of the fifty-one controls.

(b) *Urinary excretion of steroids.* The mean values for the two steroids of approximately normal distribution, 17-KS and 17-KGS, were compared in the patient and control groups. This showed there to be a significantly lower value for 17-KS in the patient group (difference $= 1{\cdot}4 \pm 0{\cdot}6$ mg/24 hr), and a non-significant tendency in the same direction for 17-KGS (difference $= 0{\cdot}8 \pm 0{\cdot}8$ mg/24 hr). The number of persons with extremely low steroid excretion was generally higher among patients than among controls, but significant differences were demonstrable only as regards TZR and DHIA. A low excretion of TZR was found in eleven of thirty-seven patients and in six of thirty-seven controls. The corresponding figures for DHIA were thirty-six of sixty patients, and twenty-eight of sixty controls.

Comparative studies of patient and control pairs showed, finally, that the values for all the steroid fractions studied were more often lower in the patients than in their controls. This tendency was significant only as regards TZR, the values in patients being lower than in the matched controls in twenty-five of thirty-six pairs.

It can be said, then, that the results of this investigation show a consistent trend, in that the excretion of steroids in the urine was generally lower in the patients than in their controls.

DISCUSSION

The results arrived at in this section agree in many respects with those of other authors. The high incidence of persons with a high degree of leadership among the patients has already been commented.

The preponderance of persons with a high fat factor among the patients conforms with the views held widely as to the physique of post-coronary patients. It should,

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however, be noted that various authors have not always drawn any clear distinction between high body-weight and the presence of excessive body-fat. In their study on the young adult male with a history of myocardial infarction, GERTLER and WHITE (1954) used among others SHELDON's classification of body-build. They found a significant predominance in the post-coronary group of endomorphic mesomorphs, (fat, muscular persons), but markedly low incidence of ectomorphs (lean persons). These authors, then, believe their series *also* to have evidenced a high muscle factor; this was not definitely verified in the present series.

The diverging results in our own study and in that cited above as regards the muscularity of the patients may (in part or wholly) be dependent upon the differing methods of determination.

SHELDON's estimation of muscularity is based upon the variable mesomorphy. However, as was shown by LINDEGÅRD (1953), there is some not inconsiderable overlapping between endomorphy and mesomorphy, in that both variables are the expression of fatness as well as muscularity. This implies that the high degree of mesomorphy in post-coronary patients reported by GERTLER *et al.* (1954) and SPAIN *et al.* (1953) may be the expression of the higher fat factor in these patients.

On the other hand, the dynamometric determination of muscularity used in the present investigation is less suitable for use in a series of post-coronary patients. As mentioned earlier, we frequently refrained from asking a patient with coronary sclerosis to exert maximal strength on dynamometric determination, in view of the danger this entailed. The mean dynamometric readings in the patient and control groups show no consistent trend towards higher values in either group. In view of the comments above, this might be interpreted as suggesting that muscularity was, in fact, higher in the infarction group. This in turn might be taken as suggestive of agreement with the findings of GERTLER *et al.* and SPAIN *et al.*, on the assumption that the high degree of mesomorphy in those studies really represented a high degree of muscularity.

Comparison of our own results with those of other authors as regards the psychologic traits peculiar to patients with a history of myocardial infarction is, of course, rendered difficult by the fact that the variables studied are not directly comparable.

The psychologic variables studied in the present investigation are best compared with those in a study by MILES *et al.* (1954). MILES *et al.* studied a group of myocardial infarction survivors and a corresponding control group; a number of personality variables were rated, and some personality tests performed. Our series, as also that of MILES *et al.* showed there to be only insignificant differences in personality pattern between the postcoronary patient and a comparable control. MILES *et al.* found a tendency towards lower powers of introspection and a difficulty in controlling aggressivity in the patients. Our series, and our methods, failed to demonstrate the presence of any such trend.

The finding that spontaneous periodic depressions are more common in myocardial infarction patients than in comparable controls appears not to have been demonstrated earlier. It is not clear whether this tendency is habitual to the patient or whether it should be regarded as a symptom of the disease or of the underlying atherosclerosis. On compiling the history, the examiner attempted as far as possible to ascertain whether traits noted were part of the patient's habitual personality pattern, dating from considerably before the onset of the acute myocardial infarction. But there was, of course, no guarantee that the interviewer could disregard current personality dimensions.

The predominance of persons with a high serum cholesterol level among the patients, as also the finding of a normal level in many patients, agrees with reports on recent studies of other post-coronary series (LERMAN *et al.*, 1946; GOFMAN *et al.*, 1950; GERTLER *et al.*, 1950; BARR *et al.*, 1951; STEINER *et al.*, 1952; SCHETTLER *et al.*, 1955; NIKKILÄ, 1955; BIÖRCK *et al.*, 1956 and others).

GERTLER and WHITE (1954) determined the urinary excretion of neutral 17-ketosteroids in eighty-seven cases in their series of young male post-coronary patients; they, as did we, used CALLOW's modification of ZIMMERMAN's technique. They found the mean value for 17-ketosteroid excretion to be lower, though not significantly so, in the patients than in the controls; the controls, however, were specially selected as regards occupation. As pointed out by GERTLER and WHITE, this non-significant difference might be accounted for by the fact that the controls were on the average 10 years younger than the patients. Recently, MARMORSTON *et al.* (1957) published a study on a series of postcoronary patients of different ages; the controls were patients in whom clinical examination of the heart revealed nothing abnormal and whose electrocardiograms were normal. The controls showed no signs of disease of the liver or kidneys or of any chronic disease whatsoever, nor were they suffering from diabetes, myxoedema, or thyrotoxicosis. In the post-coronary patients aged 55 or less, the 17-ketosteroid values were statistically lower than in the controls. We found the same tendency—also significant—towards lower 17-ketosteroid values in the patients in our series, in which the controls were more expediently selected. However, the excretion of 17-ketosteroids represents only *one* aspect of the function of the steroid-producing organs, which is better studied by determining the excretion of several steroid metabolites. In a series of unselected myocardial infarction patients in Malmö, FORSSMAN (1954) found a general decrease in the urinary excretion of 17-ketosteroids in male patients during the third to sixth post-infarction weeks (6·6 mg/24 hr as against the estimated 7·8 mg/24 hr in normal males of corresponding age); this contrasted with the significant rise during the first week after the onset. This series comprised patients of all ages. Taken together with our results, these studies show the steroid excretion of post-coronary patients after the immediately post-infarction period to remain at a lower level than that of comparable controls.

To sum up. Comparative studies of patients and controls showed the patient group to be characterized by the following features:

(1) Predominance of the occupations calling for a high degree of leadership.
(2) Higher parental mortality due to atherosclerotic cardiovascular disease before the age of 71 years.
(3) Higher incidence of excessive body fat.
(4) Higher incidence of periodic depressions.
(5) Higher incidence of hypercholesterolaemia.
(6) Lower average excretion of steroids in the urine.

STUDIES OF SUB-GROUPS AMONG PATIENTS AND CONTROLS

Comparative studies of the two groups, patients and controls, revealed dissimilarities as regards a number of variables.

On evaluating the connexion between these group differences, the question arises: Is any one set of patients chiefly responsible for all the group differences, or are some

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of the dissimilarities attributable to a certain number of patients and other dissimilarities to another? This query could also be put as follows: Are obesity, hypercholesterolaemia, and low steroid excretion found in *the same patients* or are *different patients* responsible for each of these separate mean group differences between patients and controls? Is there in the patient group a predominance of a consistent type of person as regards physical and mental make-up, or is it possible to distinguish different categories or 'types' among the patients?

We attempted to elucidate these questions in the part of the investigation reported below, by studying sub-groups of patients and controls. In this study, we used the methods described under (2) and (3) in 'Statistical methods' for both the relevant sub-group of patients (or controls) and for the rest of the patients (or controls). First, we selected sub-groups of patients—and, if possible of controls—showing extreme values for the variables for which there was a group difference between patients and controls. This give six different sub-groups, characterized by:

High degree of occupational leadership.

Death of one or both parents due to atherosclerotic cardiovascular disease before the age of 71 ('familial atherosclerosis').

High fat factor.

Presence of tendency to periodic depressions.

Hypercholesterolaemia.

Low excretion of steroids in the urine.

These studies of subsidiary groups among patients and controls were extended to embrace variables other than those in which there were group differences between the two series. Both patients and controls with a large length factor as well as those with a large sturdiness factor of the skeleton were examined. These two constitutional variables are but little affected by environmental factors (v. VERSCHUER, 1954; LINDEGÅRD, 1957), which is of interest when considering the genetic influence in myocardial infarction.

During the study of the sub-groups characterized by 'familial atherosclerosis' and by high body-weight, results were arrived at which gave reason to study in detail the sub-group characterized by the psychologic variable 'explosivity'.

Further, differences between patients in whom varying periods had elapsed between the onset of the acute myocardial infarction and the examination were studied. One might suppose the patients in cases in which a long period had elapsed to differ in many respects from those whose onset was recent. The first category of patient has had a long time in which to adapt himself to new conditions attendant upon myocardial infarction.

Earlier investigators (FELDMAN and MORRISON, 1951; DUNBAR, 1948, 1954; CURTIUS, 1954; and others) have discussed mutual and differing traits in the individual make-up of persons with peptic ulcer and with hypertensive cardiovascular disease. For that reason we carried out a separate investigation as to the way in which persons with a history of peptic ulcer, in both the patient and control groups, differed from other subjects.

For convenience, the results are presented in the order below, since they fall naturally into clusters of groups.

Social and familial:	High degree of leadership (A)
	Familial atherosclerosis (B)
Time factor:	Long interval between the acute onset of infarction and the examination (C)
Somatologic:	Large length factor (D)
	Large sturdiness factor (E)
	High degree of fat (F)
	High body-weight (G)
Psychologic:	Presence of explosivity (H)
	Disposition to periodic depressions (J)
Biochemical:	Hypercholesterolaemia (K)
	Low excretion of steroid metabolities in the urine (L)
Miscellaneous:	Presence, or history, of gastric or duodenal ulcer (M)

A. Patients with a high degree of leadership

In the occupational leader category there were fourteen patients but only seven controls. Owing to the smallness of the latter number, no sub-group of persons in the highest grade of leadership was selected for study from the controls.

Of the fourteen patients in the highest grade of leadership, twelve were classed as 'white collar workers', whereas only twelve of the remaining thirty-six patients were assigned to that category. As was to be expected, then, the degree of leadership was in inverse proportion to the degree of occupational physical activity.

The intellectual powers of the patients were significantly higher in those with a high degree of leadership than among the others. A tendency towards a higher degree of validity in patients with leadership was also noted. Both these trends may be due to the selection underlying the social distribution. A corresponding interrelation should *a priori* be revealed on examination of a control series.

B. Patients with familial atherosclerosis

The sub-group of 'persons with familial atherosclerosis' comprised seventeen of sixty-two patients, and eight of sixty-two matched controls.

Patients with familial atherosclerosis showed, on comparison with other patients, the following group characteristics:

Higher degree of capacity.

Lower degree of stability.

Non-significant tendency to a lower degree of validity.

Higher incidence of explosivity.

Tendency to a higher excretion of steroid metabolites in the urine.

The difference between patients with familial atherosclerosis and other patients emerged most clearly on comparison of the patients and their matched control. Thus, the degree of capacity of the patient belonging to this sub-group exceeded that of the matched control in eight cases, and was less than the control's in three cases; the corresponding figures for the remaining patients were twelve and twelve, respectively.

The degree of stability of the patient with familial atherosclerosis exceeded that of his matched control in only two cases, but was less in nine cases; the corresponding figures for the remaining patients were nineteen and seventeen.

The degree of validity, finally, was higher in the patient with familial atherosclerosis than in his control in four cases, and less than that of the control in ten cases; the corresponding figures for the remaining patients were nineteen and fifteen.

Explosivity was noted in nine of sixteen familial atherosclerosis patients, while this variable was recorded in 10 of the remaining thirty-nine patients.

As regards the urinary excretion of steroid metabolites, the following may be noted:

Comparative study of patients with familial atherosclerosis and their matched controls revealed in the patients a significant tendency to higher steroid values for the NF-fraction, and a non-significant tendency in the same direction for the remaining fractions (with the exception of TZR which was determined in only a small number of cases). These results, then, conflict with the findings of comparative studies of the patient group *as a whole* and with the control group as a whole (see p. 102).

C. Patients with a long period of survival between the acute onset of infarction and the examination

One sub-group (thirty members) comprised the patients whose acute onset had occurred at most 18 months before the present examination; the other sub-group (thirty-six members) was composed of the remaining patients. The period of 18 months was chosen arbitrarily in order to give groups of approximately the same size. If there were a large number of 'border cases' immediately above or below this limit of 18 months, a source of error due to chance might give rise to an illusory correlation between the individual make-up and the period of survival. However, this source of error appears not to have played any great part in our series. In only seven of the thirty cases of short survival was the interval between onset and examination from 12 to 18 months, and in only seven of the thirty-six cases in the long survival sub-group had the onset occurred from 19 to 30 months previously.

The patients of long survival differed from those whose onset had occurred within 18 months or less in evidencing the following features:

Lower body-weight.

Higher incidence of periodic depressions.

Lower sturdiness factor.

Lower degree of validity.

The sturdiness factor, recorded as the breadth across the femoral condyles, exceeded that of the matched control in only nine patients of long survival, while it was less than that of the control in twenty-two cases in the same sub-group. The corresponding figures for the patients of short survival were fourteen and seven, respectively. The ratio of high to low sturdiness factor was 8 : 15 in patients of long survival, and 11 : 5 in patients of short survival.

The body-weight was higher in the patient than in the matched control in thirteen cases and lower in eighteen cases of long survival. The corresponding figures for patients of short survival were sixteen and six respectively.

The lower body-weight of patients of long survival was accompanied by a lower sturdiness factor, and cannot, therefore, be accounted for solely by a decrease in body fat and a loss of muscle volume.

Since the sturdiness factor may be regarded as relatively constant in the individual during the course of the infarction disease, the result of this study implies that there is a significant correlation between the period of survival and a somatic factor which is constant in the adult individual. However, no conclusions as to the correlation between the soft tissues factors and the period of survival may be based on the present series, owing to inter-individual changes which can be assumed to have taken place during the period of survival itself.

A tendency to periodic depression was noted in eight of the seventeen cases of long survival, but in only three of nineteen patients of short survival. A low degree of validity (sub-validity) was found in twelve of thirty-two patients of long survival, but in only two of twenty-three cases of short survival.

Since the personality pattern may be changed by the infarction disease itself and during convalescence, the psychologic dissimilarities noted between the two sub-groups cannot be taken with certainty to reflect differences in habitual personality pattern. Both the depressions and the low degree of validity might equally well be symptoms of the disease and the limitations imposed upon the patient's general social activity by the subsequent convalescence.

D. Patients and controls with a high length factor

The length of both the tibia and the radius served as measures of the length factor in the present study. The whole series—patients and controls—was divided into three categories according to the recorded length of each of these bones; as mentioned earlier, these categories were termed 'high', 'medium', and 'low' variants. The final placing of each person in the various length-factor sub-groups was subject to the following considerations: when both the tibial and radial lengths were high, or when either one of these dimensions was high and the other medium, the subject was placed in the sub-group 'high length factor'.

Earlier studies have demonstrated a positive correlation between the magnitude of the length and sturdiness factors (LINDEGÅRD, 1953, 1956). A similar tendency was noted in the present investigation in both patients and controls; however, this relation was significant only in the control series. Equally, a tendency to co-variation between the length factor and body-length was verified in both patient and control series.

The patients with a high length factor differed from the remaining patients in the following additional respects:

Tendency to a higher degree of stability.

Higher degree of solidity.

Higher serum cholesterol level.

Controls with a high length factor were found to have a corresponding tendency to a higher degree of stability and a higher cholesterol level. There was no single instance of hypercholesterolaemia among twenty-four controls with a high length factor. On the other hand, there were eight cases of hypercholesterolaemia among forty-eight controls with medium or low length factors. These figures comprise not only the matched controls, but also those selected for the patients with signs of atherosclerosis other than myocardial infarction. This difference between controls with a high length factor and those with medium or low length factors was statistically significant ($p = 0{\cdot}03$).

E. Patients and controls with a high sturdiness factor

As mentioned earlier, the sturdiness factor was gauged by the breadth across the femoral condyles and the bi-malleolar width. Classification into sub-groups of high, medium, and low variants was analogous with that of the length factor described in the preceding section.

In this, as in earlier studies, both the patients and controls with a high sturdiness

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factor showed a parallel tendency to high length and muscle factors, longer body-length, and greater body-weight.

In both the patients and controls in the present series, there was a tendency to interrelation between the sturdiness and fat factors. In the control series, two of the skin-fold dimensions showed a significant co-variation with the sturdiness factor. A similar tendency was observed in the patient series, but it was not significant. This finding is worthy of note; it has frequently been claimed that if there were any correlation between the fat factor and the other body-build dimensions, it would consist in an inverse correlation between the fat factor and the sturdiness or length factors. In both KRETSCHMER's and SHELDON's systems of differential somatology, body fat and sturdiness are regarded as a cluster in which a high degree of body fat co-exists with a low sturdiness factor, termed respectively by the authors the pyknic and dominant endomorphic type. Since LINDEGÅRD's system of differential somatology deals separately with each somatologic variable, it is now possible to study in detail the interrelation between the individual variables. The correlation postulated by KRETSCHMER and SHELDON was not verified with LINDEGÅRD's system, at least not in the present series. On the contrary, a tendency in the opposite direction was noted, as already mentioned.

In the control series, the sturdiness factor and the degree of validity were correlated, in that a high sturdiness factor was frequently associated with a high degree of Validity. A non-significant tendency in the same direction was noted in the patient series. This latter relation may, however, have been the result of the influence upon the interviewer of a halo effect connected with the observation of the subject. Consequently, no great import can be read into this finding.

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F. Patients and controls with a high fat factor

Both patients and controls with a high fat factor were heavier in body-weight than the remaining persons in the two groups. No definite correlation was otherwise demonstrable between the fat factor and the other somatologic variables studied.

In both patients and controls, the fat factor was found to be related to the two psychologic variables, Stability and Validity. Persons with a high fat factor generally showed a higher degree of Validity and a lower degree of Stability. Both these findings are, however, subject in unknown measure to a halo effect deriving from the interviewer's observation of the subject. Consequently, the findings permit of no statistic analysis.

Patients with a high fat factor appeared on the average to show a higher incidence of hypercholesterolaemia than others. Comparison of the patient and his matched control showed the patient's serum cholesterol level to exceed that of his control in nineteen of twenty-four pairs in which the patient had a high fat factor, while the corresponding figures for the rest of the patients were fifteen of twenty-eight pairs. The difference was significant.

G. Patients and controls with high body-weight

Since body-weight is a collective measure of different tissue components, it may be expected to be correlated with the various somatologic variables recorded. Both patients and controls with high body-weight were in fact found to differ from the remaining in that the size of the length, sturdiness, muscle, and fat factors, and also body-length was larger.

Just as other somatologic factors were found to be related to the psychologic variables, body-weight showed correlation with validity, stability, and solidity. In the patient group, persons with high body-weight were found to differ significantly from others in their higher degree of validity, lower degree of stability, and higher degree of solidity. In the controls, such a tendency was statistically verifiable only as regards validity.

In the patient group, two of the discontinuous psychologic variables were found to be related to body-weight. Patients with high body-weight showed a higher incidence of explosivity and a tendency to a lower incidence of impairment than patients of medium or low body-weight.

Body-weight was also found to be correlated with the biochemical variables in the patients. Thus patients with high body-weight differed from the others in their tendency to a higher incidence of hypercholesterolaemia and an increased urinary excretion of steroids.

The serum cholesterol level in patients of high body-weight exceeded that of their matched controls in sixteen of nineteen pairs, while the corresponding figures for the remaining patients were eighteen of thirty-one. The mean urinary excretion of steroids exceeded that of the matched control in thirteen of twenty-one patients of high body-weight, whereas in the remaining patients this value was higher than that of the matched control in only nine of thirty-four pairs. This difference was highly significant. Among the individual steroid fractions, it was chiefly the 17-ketosteroidogenic steroids which showed the same tendency to higher values in patients of high body-weight, although a similar trend was noted for the neutral 17-ketosteroids, the non-acid fraction, and dehydro*iso*androsterone.

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The interrelation between body-weight and urinary steroid excretion was demonstrated by LINDEGÅRD and NYMAN (1956) in a series of Swedish Army men. In that series, the correlation was most marked between the muscle factor and steroid excretion. In the present series, there was no demonstrable correlation between the length, sturdiness, and fat factors on the one hand, and the excretion of steroid fractions on the other. If, however, the significant correlation between body-weight and steroid excretion is taken as the basis for a purely numerical analysis of the relationship between the different somatologic variables and steroid excretion, the following emerges. In patients with high length and sturdiness factors, the steroid excretion exceeded that of the controls as often as it fell short of the control level. In patients with medium or low length and/or sturdiness factors, however, the level of steroid excretion was usually less than that of the matched control. This shows, then, that the relation of the length and sturdiness factors to steroid excretion is in principle the same as that of body-weight to steroid excretion. On the other hand, the fat factor differs from the two somatologic variables mentioned above in lacking even numerical evidence of any relationship with steroid excretion. The muscle factor was not included in this analysis in view of the objections which may be raised as regards the estimation of this somatologic variable. The results obtained may be interpreted as implying that the correlation of body-weight with steroid excretion derives largely from the non-fatty body tissues.

The tendency to correlation between the serum cholesterol level and body-weight was also found between the serum cholesterol level and the fat as well as the length factor. There is, then, no differentiation between the fatty and non-fatty body tissues as regards their correlation with serum cholesterol.

H. Patients and controls with Explosivity

Comparative studies of individuals with explosivity regarding other dimensions of the individual personality pattern revealed a number of differences between patients and controls.

The relationship between explosivity and familial atherosclerosis was not analysed in the controls, since very few reported the death of one or both parents before the age of 71 from cardiovascular atherosclerotic disease. However, among the patients, familial atherosclerosis was found in nine of nineteen explosive patients as against eight of thirty-nine non-explosive patients. The difference was significant. The patients with explosivity differed from those lacking this feature in that they had a higher body-weight than their matched controls. This higher body-weight appeared to be due chiefly to higher sturdiness and muscle factors, and scarcely at all to the length and fat factors.

The body-weight of the explosive patients exceeded that of their controls in thirteen of eighteen pairs, whereas only fifteen of thirty-five non-explosive patients were heavier than their matched controls.

In the control group, there was no evidence whatsoever of any tendency to higher body-weight in explosive than in non-explosive persons.

As regards the continuous psychologic variables, the presence or absence in patients of explosivity was associated with a significant difference in the degree of stability.

The degree of stability in explosive patients was lower than in the matched control in twelve of fourteen pairs, while it exceeded that of the matched control in nineteen of thirty-three non-explosive patients. Superstability was noted in only three of the explosive patients as against fifteen of the non-explosive. The corresponding figures for substability were twelve in the explosive and nine in the non-explosive sub-groups. No trace of this relationship between sub-stability and explosivity was found among the controls.

As regards the discontinuous psychologic variables, the presence of explosivity was found to be associated chiefly with the presence of sensitivity to injustice, and to a lesser degree with the presence of viscosity. This association was found in both patients and controls and cannot therefore be taken as specific to myocardial infarction patients.

Explosive and non-explosive patients showed a tendency to differ as regards the urinary excretion of steroids. Collectively the patient series showed a lower excretion of steroids than the matched controls, but on being divided into explosive and non-explosive sub-groups a certain differentiation was noted. The steroid excretion in explosive patients was found equally often to exceed as to fall short of the level in the matched controls, whereas in patients lacking explosivity the tendency was similar to that in the series as a whole—steroid excretion as a rule lower in the patient than in the control. This difference between explosive and non-explosive patients was statistically significant only as regards one steroid fraction, the non-acid fraction. (The value in the explosive patient exceeded that of the matched control in twelve of nineteen pairs as against eleven of thirty-eight non-explosive patients.)

No such correlation emerged on analysis of the control group, not even as regards the numerical tendencies.

Thus, patients with explosivity differed from the controls regarding many of the

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variables studied. Contrary to the controls, patients belonging to this sub-group were characterized by a heavy, voluminous body, a sub-stable personality, and relatively high urinary excretion of steroids. In addition, the incidence of familial atherosclerosis was higher in the patients with Explosivity than in patients in whom this feature was lacking.

This suggests that a special category may be distinguished among the patients, characterized by explosivity among other features; this category seems to be less frequent among the controls.

J. Patients with a tendency to periodic depression

Since this trait was noted in only three of the controls, no separate analysis was made in the control series. The tendency to periodic depression was also fairly rare in the patient group, being recorded in only eleven cases. Consequently, the results of this analysis should be interpreted with caution.

Patients suffering from periodic depression did not differ from others as regards somatologic or biochemical variables. On the other hand, a significant dissimilarity between these and other patients was noted as regards the degree of stability and the presence of hypersensitivity to injustice.

Patients with periodic depressions were more commonly sub-stable and hypersensitive to injustice than the other patients.

The degree of stability in patients with periodic depression was lower than that of the matched control in seven of eight pairs, while in the remaining patients stability was lower in patient than control in only eighteen of thirty-eight cases. Super-stability was noted in one of the patients with periodic depressions, as against seven instances of substability; the corresponding figures in patients not suffering from periodic depression were seventeen and fourteen. Sensitivity to injustice was recorded in seven of eleven patients with a tendency to periodic depression, but in only five of the remaining twenty-four patients.

The tendency to periodic depression cannot with certainty be regarded as a habitual personality trait, it may be superimposed by the atherosclerotic disease, the infarction, or the subsequent convalescence. The association of this tendency to periodic depression with sub-stability may also be a secondary phenomenon.

K. Patients with hypercholesterolaemia

Owing to the small number of persons with hypercholesterolaemia in the control series, only the patients were analysed in this respect.

There was a highly significant difference as regards body-weight between hypercholesterolaemic patients and those not showing a high serum cholesterol level. Hypercholesterolaemia and high body-weight appear to be associated.

In the hypercholesterolaemic sub-group, the patient's body-weight exceeded that of his matched control in thirteen of fifteen pairs, whereas in the remaining patients body-weight was greater than that of the control in only eleven of twenty-eight pairs. The ratio of high to low body-weight in the hypercholesterolaemic sub-group of patients was 9 : 1, and in the remaining patients 10 : 8.

If this finding is compared with the correlation shown between hypercholesterolaemia and the different somatologic variables expressing the quantity and distribution of bone, muscle, and fatty tissues, it emerges that with the exception of the fat factor all the recorded variables reflect the tendency shown in relation to body-weight. Thus, patients with hypercholesterolaemia tend to higher length, sturdiness,

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and muscle factors than non-hypercholesterolaemic patients, but not a higher fat factor.

This lack of interrelation between hypercholesterolaemia and the fat factor does not agree with the results of the comparative studies on the separate variables in relation to the serum cholesterol level, which showed the factor to be correlated with the cholesterol level. Thus, patients with a high fat factor showed, as sub-group, hypercholesterolaemia.

Of the four continuous psychologic variables studied, Capacity was foremost in showing a highly significant negative correlation with the serum cholesterol level. Hypercholesterolaemic patients had a lower degree of capacity than their matched controls. (The degree of capacity of hypercholesterolaemic patients exceeded that of the control in two of ten pairs; among the remaining patients, the patient's degree of capacity was higher than the control's in fourteen of nineteen cases.)

Dissimilarities were noted between hypercholesterolaemic and non-hypercholesterolaemic patients as regards stability and validity. The former category of patient tended to a higher degree of validity and a lower degree of stability. In neither instance was the tendency statistically significant.

There was no differentiation between hypercholesterolaemic and non-hypercholesterolaemic patients as regards the discontinuous psychologic variables.

It should be noted in this connexion that the interviewer was not informed as to whether the subject belonged to the patient or control series when rating personality. By witholding this information, the halo effect deriving from his preconceived notions was eliminated. Therefore, the differences noted in psychologic dimensions between hypercholesterolaemic and non-hypercholesterolaemic patients cannot be attributed to any halo effect. But there still remains the question as to whether the differences noted were primary deviations relating to the habitual personality patterns of the subjects, or whether they should be interpreted as secondary changes in personality resulting from the disease. Both the low capacity and the tendency to a low degree of stability might be attributed to early aging brought about by the atherosclerotic disease. In that event, however, persons with the impairment and possibly with the periodic depressions associated with atherosclerotic cerebral lesions would have predominated. The absence of any preponderance of such persons suggests that the deviations noted represent truly habitual differences in personality between the two sub-groups of patients.

The hypercholesterolaemic patients differed from the others as regards the urinary excretion of steroids. This is worthy of special note. As mentioned earlier, the whole myocardial infarction series was characterized by lower steroid excretion than the matched controls. In the hypercholesterolaemic sub-group of patients, on the other hand, the steroid excretion was as high or in some instances even higher than that of the matched controls.* This tendency was most striking as regards the collective mean value calculated for all the steroid fractions determined. Among the individual fractions, the neutral 17-ketosteroids showed a statistically significant difference.

The collective mean excretion of steroids in the urine exceeded that of the matched control in nine of thirteen patients with hypercholesterolaemia, whereas the patient level exceeded that of the

* In this connexion it should be mentioned that evidence is now at hand that cholesterol and particularly acetate are precursors of the steroid hormones (BLOCK 1945, ZAFFARONI *et al.*, 1951, BRADY, 1951, SAVARD *et al.*, 1952, UNGAR *et al.*, 1953).

control in seven of thirty non-hypercholesterolaemic patients. The difference was highly significant. With the exception of the tetrazoleum-reducing fraction, all the steroids determined showed the same tendency, that is to higher values in the hypercholesterolaemic sub-group than in the controls. However, the tendency was statistically verified only as regards the neutral 17-ketosteroids. The incidence of extreme variants showed a highly significant difference between the two sub-groups of patients. A low excretion of 17-ketosteroids occurred in only three of the sixteen hypercholesterolaemic patients, while the corresponding figures for the remaining patients were no less than seventeen of thirty. A tendency in the same direction was noted for all the other fractions determined, including the tetrazoleum-reducing fraction.

L. Patients and controls with low urinary steroid excretion

There was a tendency to a low incidence of leaders among persons showing a low steroid excretion in both the patient and control series. This tendency was significant in the control series, but not statistically verified in the patient series. There was a parallel tendency in both patients and controls to a higher degree of occupational physical activity among persons with low steroid excretion. However, this tendency was significant in neither series.

With the exception of the correlation in the patient series between steroid excretion and body-length (low steroid excretion usually being associated with short body-length), no interrelation was demonstrable between steroid excretion and somatologic variables.

As when basing analysis upon the serum cholesterol level, classification according to steroid excretion showed a negative relationship to exist between steroid excretion and serum cholesterol level. This association leads to a low incidence of persons with a high serum cholesterol level among patients with low steroid excretion.

In fact, there were only four patients with hypercholesterolaemia among twenty-three with low steroid excretion. On the other hand, there were as many as thirteen hypercholesterolaemic patients among twenty-seven in whom steroid excretion was not low. This difference was significant.

We found, then, a negative correlation to exist between steroid excretion and serum cholesterol level in patients but not in controls. The higher the serum cholesterol level, the lower the steroid excretion, and vice versa. But this correlation was not verified in the control series. These findings will be discussed at more length in Section 8.

M. Patients and controls with a history of peptic ulcer

It has been said (KAUFMANN, 1943, WRETMARK, 1953, and others) that the individual make up of patients with peptic ulcer differs from that of comparable controls. This applies to both physical and mental traits. Peptic ulcer and myocardial infarction have frequently been attributed to common aetiologic factors, chiefly to severe mental stress. Consequently, special attention was given in the present study to the relationship between peptic ulcer and myocardial infarction.

Among the sixty-two patients, there were eleven with a history of peptic ulcer. For the diagnosis to be accepted, it was necessary that the patient had been informed by his physician after roentgenographical examination that he was suffering from gastric or duodenal ulcer. There were also eleven instances of peptic ulcer in the sixty-two matched controls. The incidence of peptic ulcer was, then, identical in the two series. This agrees with the statement made by WALSH (1941) to the effect that there is no difference in the incidence of peptic ulcer in post-coronary patients and comparable

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controls. On the other hand, FELDMAN *et al.* (1951) and others have expressed the view that ulcer is somewhat more common in post-coronary patients than in controls.

As regards somatologic features, various authors (KAUFMANN, WRETMARK, and others) have shown body-volume to be smaller in relation to body-length in persons with ulcer than in controls. A similar trend was noted in the present investigation. Among the patients with ulcer, persons of high body-weight were significantly fewer than among the other postcoronary patients. This appeared to be due chiefly to a lower degree of fat among the postcoronary patients with ulcer.

WRETMARK, in his study of 1953, used in part the same methods of recording the psychologic exploration as were applied in the present investigation. The results, therefore, are comparable in some measure. In the presence of ulcer, WRETMARK found a lower degree of validity and higher degree of stability, together with a tendency to higher capacity and lower solidity, than in the controls. We have failed to verify these findings statistically in the present study. But it should be borne in mind that the number of persons with peptic ulcer in both our patient and control groups was very small. In our control series, a tendency to a lower degree of Solidity was noted in persons with ulcer. This difference was significant.

In WRETMARK's study, stability appeared to be the personality dimension most closely related to the ulcer condition, as such, and to its prognosis. It may be of interest to note that in our ulcer cases in the control group, superstability was recorded in six cases as against normo-stability or sub-stability in five cases. The tendency, then, was similar to that reported by WRETMARK. However, among the patients with ulcer, superstability was noted in only one of the ten cases explored as to personality dimensions. This result shows superstability to be significantly ($p = 0{\cdot}04$) less common in persons with ulcer coexisting with myocardial infarction than in the ulcer cases among the controls.

The degree of capacity also showed a tendency to differ in postcoronary patients and controls with ulcer. Super-capacity was noted in only two of the ten post-coronary patients with ulcer, while no less than six of the eleven controls with ulcer were rated as supercapable. The difference was not significant in the present series.

To sum up, it may be said that earlier observations by other authors on the individual make-up of patients with peptic ulcer were in part verified. Patients and controls with ulcer tended to differ in certain respects. These differences may be interpreted as implying that the features specific to ulcer-sufferers—as described by other authors—were most striking in the controls with ulcer. The post-coronary patients with ulcer, on the other hand, showed no definite tendency to those characteristics—at least not as regards personality pattern.

DISCUSSION

Among the group differences emerging on comparative study of the patient and control series as wholes, were the high serum cholesterol level and low urinary steroid excretion of the patients. Analysis of sub-groups showed, however, that the patients with hypercholesterolaemia as a rule had *higher* urinary steroid excretion than the remaining patients. It was also noted that patients with low urinary steroid excretion only rarely had a high serum cholesterol level.

These observations justify the following conclusions: (1) the lower urinary

steroid excretion in the patient group as a whole derived chiefly from patients other than those with hypercholesterolaemia, and (2) the higher serum cholesterol level in the patient group as a whole was brought about by patients other than those with low urinary steroid excretion.

This, in turn, implies that the differences demonstrated between the whole patient and the whole control groups as regards the serum cholesterol level and urinary steroid excretion were not attributable to any single category of phenotypic patients. Instead, one must assume at least two different categories of patients to have been responsible, if any such unitary categories in fact existed in the present series. If, however, there were such categories, the results suggest that hypercholesterolaemia is a component of one of the patients' 'type' make-up, and the low steroid excretion a component of another 'type'.

These observations led us to analyse the results of the study with a view to isolating, if possible, different 'types' of post-coronary patient. Before discussing this matter further, we would like to mention some of the principles governing this 'typing' of persons.

Clearly delimited individual types are distinguishable in the literal sense only as regards discontinuous variables, such as the different blood groups. When one is dealing with continuous variables there is, of course, no natural boundary separating different categories of persons from each other. Definite *types* cannot be distinguished even by taking into consideration several continuous variables at the same time and finding that a certain category is characterized by extreme values for each of these variables. The term 'type' is, however, in general use for this purpose even when applied to continuous variation. These 'types' denote persons with a combination of extreme variants of several dimensions. This is exemplified by the use of the term *athletic type* for a person with *large* sturdiness and muscle factors together with *small* length and fat factors.

A coexistence of extreme variants of several variables, as assumed in this paper, may be either purely 'coincidental' or the result of a systematic accumulation. (This systematic accumulation may, in that event, be of genotypic and/or environmental origin.)

When the 'typical' accumulation of high or low values for different variables is due to chance variation, the number of 'types' within a population will correspond to a statistical calculation of random coincidence. When, however, there is a systematic accumulation of extreme variants, one should be justified in expecting 'types' to occur more often or more rarely than predicted by random coincidence.

Assuming the existence of a particular 'type', although there is an affinity between extreme variants of different dimensions, one or other of the components may predominate in separate individuals. If, for example, a type is characterized by a certain profile, a certain member who on the whole is assignable to that type may show extreme values for some of the variables included in the profile pattern and medium values for the rest.

With these general typologic considerations in mind, we attempted to ascertain whether our observations as regards hypercholesterolaemia and low steroid excretion could be taken to imply that there was some systematic accumulation of different extreme variants in our patient series, or whether their coexistence should be taken to be due solely to random coincidence.

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We based our analysis upon the respective features distinguishing hypercholesterolaemic patients and patients with low urinary steroid excretion from the remaining patients. The phenotypic features will be found in the following list:

Hypercholesterolaemic patients were generally characterized by:	*Patients with low urinary steroid excretion* were generally characterized by:
High or medium urinary steroid excretion	Low or medium serum cholesterol level
Heavy body-weight	Short body-length
Large length factor	
Large sturdiness factor	
Large muscle factor (probable)	
Low capacity	
High validity	
Low stability	

Statistic variation may result in a slightly different degree of affinity between variables studied if the approach is made from sub-groups of patients marked by the traits listed above as characteristic of hypercholesterolaemic patients and those with low urinary steroid excretion. Equally, these two groups of patients might also show features largely similar to quite different sub-groups not selected with regard to the characteristics specified above. For that reason we investigated the serum cholesterol level and the urinary excretion of steroids in sub-groups of patients characterized by extreme values for other variables studied. This verified several of the correlations listed above. *In addition*, a high cholesterol level was most commonly present in the following categories of patients: in those with familial atherosclerosis and with large fat factor, and in those with explosivity. These sub-groups of patients also had different combinations of traits in common with the hypercholesterolaemic sub-group of patients. Thus, patients with a large fat factor had a high degree of validity and a tendency to low stability; patients with familial atherosclerosis had a low degree of stability and a tendency to elevated urinary steroid excretion; and, finally, patients with explosivity showed a tendency to larger sturdiness and muscle factors, low stability, and a tendency to high urinary excretion of steroids.

Low urinary excretion of steroids was found as a rule in patients of light body-weight and in cases in which more than 18 months had elapsed between the onset of myocardial infarction and the examination.

As was to be expected, then, the degree of affinity between different variables was found to differ somewhat according to the angle of approach, that is, according to the sub-group analysed. The differentiation of certain patient categories must, therefore, be based on synthetic interpretation of different observations rather than on rigid statistic evidence.

To the features of patients characterized *among other things* by hypercholesterolaemia, these analyses appeared to justify the addition of the following attributes: large fat factor, history of familial atherosclerosis, and presence of explosivity. Also, the second type of patient may be further characterized by low body-weight and a relatively long period of survival (18 months or more).

On comparison, these two types of patient are found to be opposites in the majority of respects.

This raises the problem of the group differences between the entire patient and control series. The basic patient types might be such that their respective features cancel out each other on comparison of the group as a whole with the controls. This is exemplified by the variable, body-weight. The first category of patients are characterized by heavy body-weight, while the second category members are not heavy. Comparison of the patient group as a whole with the control series showed no significant difference in body weight between patients and controls. The inference is that other characteristics specific to different sub-groups of patients may fail to emerge on comparison of the two groups as wholes.

Consequently, the analysis was extended to embrace comparative studies of sub-groups of patients selected for their extreme variant of a certain dimension and *corresponding* sub-groups among the controls. This study comprised sub-groups which might be taken to be connected with the two categories of patient already distinguished. The aim of this analysis was to attempt to detect features—over and above those already distinguished—more frequently characterizing either of these patient types than the corresponding type among the controls.

Comparison of patients and controls with a large *length factor* revealed a predominance of hypercholesterolaemic persons among the patients (ten cases among twenty patients, but no instance of hypercholesterolaemia among twenty-four controls). This difference is highly significant; tall patients and controls showed no consistent tendency to differ as regards the other variables studied.

Comparative studies of patients and controls with a low or medium length factor showed no preponderance of hypercholesterolaemic patients (eight of thirty-four patients were hypercholesterolaemic, and eight of forty-eight controls). But the analysis revealed a predominance of patients with a high degree of occupational leadership and low solidity. Eleven of forty patients were high in leadership, but only five of fifty-four controls. Sub-solidity was recorded in twenty-three of thirty-eight patients, but in only seventeen of forty-nine controls. These differences did not emerge on examination of tall patients and controls.

Comparison of patients and controls with a large *sturdiness factor* showed a significant predominance of hypercholesterolaemics among the patients (ten cases in twenty-six patients, as against two in twenty-eight controls). No definite deviation in serum cholesterol level was demonstrable between patients and controls with low or medium sturdiness factors.

The urinary excretion of steroids did not differ in patients and controls with a large sturdiness factor. On the other hand, steroid excretion was lower in patients with a low or medium sturdiness factor than in the corresponding controls. These differences were significant for the non-acid steroid fraction (low excretion in nineteen of thirty-six patients as against fifteen of fifty-one controls) and for dehydro*iso*androsterone (low excretion in twenty-five of thirty-six patients and in twenty-two of fifty-one controls).

Patients and controls with a low or medium sturdiness factor differed in degree of occupational leadership. A high degree of leadership was found in eleven of thirty-six patients, as against four of fifty-two controls. This tendency was not noted among persons with a large sturdiness factor.

A high *fat factor* was found to be associated with hypercholesterolaemia in eleven of twenty-six patients, but in only two of eighteen controls. This difference is significant. There was also a predominance among the patients of persons with a high degree of capacity (seventeen of twenty-eight patients were super-capable, but only five of twenty-two controls), and a low degree of validity (seven of twenty-seven patients were sub-valid, but none of the twenty-two controls). Both these differences are highly significant. None of these three deviations between patients and controls were demonstrable in persons with a low or medium fat factor.

Classification into sub-groups according to *body-weight* showed there to be a highly significant predominance of hypercholesterolaemics among patients of high body-weight (nine of twenty patients had hypercholesterolaemia, as against only one of twenty-three controls). No such tendency was noted in the other weight categories.

Patients of medium or low body-weight had a high proportion of leaders not noted in the heavy

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body-weight category (nine leaders among thirty-nine patients, as against five leaders among sixty-one controls). The difference is significant. There was also a smaller number of super-solid persons among the patients than among the controls of low or medium weight (two super-solid persons in thirty-three patients, as compared with eighteen in fifty-five controls).

Comparison of patients and controls with explosivity showed a clear predominance among the patients of persons with a high fat factor and heavy body-weight. The high fat factor was most clearly demonstrated—being highly significant—by the parathoracic skinfold measurement (fourteen of nineteen patients had high values, and nine of thirty-three controls). A significant tendency in the same direction was noted for the subscapular skinfold thickness. Persons of heavy body-weight numbered twelve of eighteen patients, and six of twenty-nine controls. The difference is significant. No such deviation between patients and controls was noted in the non-explosive category.

Comparative studies of patients and controls with low urinary excretion of steroids showed the two groups to differ only in capacity. The patients were high in capacity, there being fifteen super-capable patients among twenty-four, as against only six among nineteen controls. The difference is significant. Patients and controls with high or medium urinary steroid excretion differed as regards the fat factor; persons with a high fat factor predominated among the patients. This was highly significant as regards the subscapular skinfold thickness, and significant when measured parathoracically.

The results of these comparative studies of sub-groups of patients with extreme values for a certain variable and *corresponding* sub-groups of controls, showed that to the features of patients characterized by low steroid excretion among other things it was justifiable to add the following attributes:

High degree of leadership	High capacity
Small length factor	Low solidity
Light body-weight	

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The features of the first type (characterized among other things by hyper-cholesterolaemia) appeared to be more consistent than those of the second type (characterized among other things by low steroid excretion). However, certain less consistent combinations of traits were also found in the first type. Hypercholesterolaemic patients were marked by low capacity and high validity. These traits were not perceptible when exploring the supposed type from other approaches. The fat factor may be taken as an example: patients with a high fat factor were characterized by high capacity and relatively low validity. Since these are psychologic variables and rated subjectively, the influence of a halo effect cannot be ruled out. Consequently, capacity and validity were excluded from the characteristics listed above under the two types.

Thus, the comparative studies reported above verified the existence of two fairly consistent types of patient. These two types were characterized by the following traits:

Type I	*Type II*
Hypercholesterolaemia	Low or medium serum cholesterol level
High or medium urinary steroid excretion	Low urinary excretion of steroids
History of familial atherosclerosis	High degree of occupational leadership
Presence of explosivity	Low solidity
Low stability	(?) High capacity
Large length factor	Small length factor
Large sturdiness factor	Small sturdiness factor
Large muscle factor	Light body-weight
Large fat factor	Period of survival exceeding 18 months*
Heavy body-weight	

* That is, the period elapsing between the onset of myocardial infarction and the examination.

Type I and type II have been evolved by synthetic interpretation of individual findings compiled in the tables in the appendix. If the table results are compared with the features distinguishing type I and type II, they will be found to fit in very well with the synthetic interpretation upon which the characterization of type I and type II patients is based.

It cannot be stressed too strongly that the description of the two types of patient given above refers *exclusively to the phenotype*. In the following section, the existence of genotypic features is discussed.

COMMENTS

Many comparative studies of post-coronary patients and normal 'healthy' controls have been conducted. The results of different investigations, however, diverge fairly widely and are in some instances contradictory. This applies to environmental factors such as occupational or nutritional habits, and the features of mental, physical, and biochemical make-up. Having made a comprehensive multidisciplinary investigation, GERTLER and WHITE (1954) stressed that further investigations are desirable, since the relationship between the various findings is far from clearly understood. The results hitherto obtained do not allow the description of any 'typical' post-coronary patient showing most of the characteristics revealed in group comparison of patients and controls.

The results arrived at in the present study indicate strongly that there exists no unitary post-coronary type of middle-aged male patient. However, there is strong evidence pointing to the existence of post-coronary *types*, i.e. unitary categories among the patients.

Our study suggested the existence of *two main postcoronary types of patient*, between which there are considerable differences as regards mental, physical, and biochemical make-up. These types are described in detail in the foregoing in the Discussion Section. Our results were arrived at by a systematic search for correlations between the various psychologic, somatologic, and biochemical variables.

It should strongly be emphasized that the 'pure' typical forms of these two types of post-coronary patient are exceptional. Generally, the patients are more or less typical of one of the two *basic types*, some patients being intermediate between the two basic types. As is mentioned above, these basic types are termed type I and type II in the present paper.

In the present series, the distinctive type II patient was somewhat more frequently represented than the distinctive type I patient. We believe the proportion of the two basic types in a series of post-coronary patients to depend, among other things, on the principles governing the selection of the series. The varying representation of the two types in a particular series of postcoronary patients might in some measure account for the apparently contradictory results arrived at in different studies. In some series, the differences between the unselected patient group and their controls show the features of type I; in others, the dissimilarities correspond more closely to a predominance of the type II patient.

In this connexion, it should be noted that the age of the patients is a variable of probable importance. It is known that the psycho–physical make-up of the individual undergoes appreciable change with advancing age. For instance, the mean excretion of steroids in the urine in a population decreases with increasing age (HAMBURGER,

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1948; HAMILTON and HAMILTON, 1948; KIRK, 1949; MCGAVACK, 1951; HAMILTON *et al.*, 1954; PINCUS, 1956; and others). The mean serum cholesterol level for men, but not for women, begins to decrease around the age 55–60 (KEYS *et al.*, 1950; GERTLER *et al.*, 1950; SCHETTLER, 1953; NIKKILÄ, 1955; LEWIS *et al.*, 1957). In this connexion it should be mentioned that it is particularly the young coronary patients who appear to have considerably higher total cholesterol values than the controls (OLIVER *et al.*, 1953; BIÖRCK *et al.*, 1956). Body-build also undergoes change with advancing age (BÜCHI, 1950; NEWMAN, 1952; and others). The fat factor, for instance, increases from the start of adult life to the start of old age and subsequently decreases. The muscle factor increases during adolescence and during adult life up to the age of 30 to 40 years. The sturdiness factor also seems to increase slightly up to old age. The observable psychologic dimensions should also be subject to change with age.

The various changes incident to advancing age may impede the differentiation of discrete types on examination of a patient or control group of elderly persons. This is exemplified by the reports of several authors as to the very striking predominance of men in myocardial infarction series of young patients, while the representation of the two sexes is more or less equalized in older patients.

The series studied by the present authors was selected not only as regards age (maximum 55 years) but also with respect to race, sex, period of survival, and place of residence (urban population). These factors may also influence the results of the investigation. Had the composition of the series been another, the results might well have been different.

As was mentioned in the introduction to this paper. the aim of this study was chiefly to detect such phenotypic features in post-coronary patients as might permit the institution of preventative measures directed against the onset of myocardial infarction or a recurrence after the initial attack.

The phenotypic dissimilarities detected cannot, however, serve for this purpose as they stand. First, it must be decided which factors may have been responsible for the characteristics noted. Attention must be given primarily to such changes as might be *secondary to myocardial infarction.*

Restrictions in daily life and various changes incident to convalescence are known to lead to decreases in the urinary steroid excretion of patients. In patients with fractures, acute haemorrhages, and post-surgical conditions, an initial rise in 17-ketosteroids in the urine (FORBES, 1943; FORBES *et al.*, 1947; KINNUNEN *et al.*, 1951; and others) is followed by a decrease in the 17-ketosteroid level to sub-normal values and only gradually by a return to normal. FORSSMAN (1954) reported a rise in 17-ketosteroids and other urinary steroid fractions during the first 5 days following the onset of myocardial infarction, but as a rule sub-normal values during convalescence —generally from the third to sixth weeks. The depressed steroid excretion found in our series, in type II patients at least, agrees with the above observations.

Patients who have suffered myocardial infarction have in recent years been recommended dietary restrictions. This has resulted in many cases in a decrease of the fat factor to smaller values than obtained before the onset of the disease. Diminished vitality and lowered physical activity may well have led to changes in muscularity. As was shown by LINDEGÅRD (1956), restricted physical activity leads within a few months to a not inconsiderable change in muscularity.

Reactive mental changes may also occur in infarction patients. These are

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probably attributable to purely organic changes brought about by the atherosclerosis with early mental aging, and to a psychogenic evolution deriving from the infarction. This latter factor has been studied by MILES *et al.* (1954) who point out that the attack leads to a collapse of their self-image as strong and successful men, and that they usually suffer subsequently from severe and prolonged anxiety, depression, and neurotic symptoms.

Comparative studies of patients and controls in our series showed a predominance in the patient group of persons suffering from periodic depression. This trait was not specific to either of the two basic types of patients. Consequently, one might take periodic depression generally to be a symptom secondary to myocardial infarction. On the other hand, the psychologic dissimilarities noted between type I and type II patients might equally well be regarded as habitual differences existing before the onset of the disease. However, we have failed to find any evidence in support of this assumption.

At this juncture, attention should be drawn to the lack of reliability attaching to the psychologic rating. Preconceived ideas on the part of the interviewer as regards the general habit of the subject and his psychomotoricity may lead to over- or underestimation of certain qualities. The influence of such a halo effect has already been mentioned as regards certain of the psychologic differences noted between type I and type II patients. Its possible effect upon the results led us to exclude certain observations from the final characterization of the patient types.

If the observations are to serve as a basis for assessment of the danger of myocardial infarction supervening in healthy persons, consideration must also be given to the source of error deriving from the selection of the patients, in that only survivors undergo examination. In general clinical experience (WRIGHT *et al.*, 1954; and others), the mortality in myocardial infarction is greatest during the first week or weeks after the acute onset. Studying in part the same population as in the present investigation, but including all ages and both sexes, FORSSMAN (1954) found the mortality among myocardial infarction patients to be 29 per cent during the first 3 weeks after the acute onset. Of the thirty-two patients below the age of 50 years, only three succumbed during this period; the number of patients below the age of 60 years was 100, of whom thirteen died during the same period. This primary mortality in middle-aged as compared with older post-coronary patients is so low that there is no reason to suppose the results obtained in the surviving myocardial infarction patients in our series to have been affected in any great measure by the selection of the patients in this respect.

The high proportion of persons with familial atherosclerosis in the patient group as a whole, and among the type I patients in particular, suggests genetic factors to be of importance in myocardial infarction. However, a history of familial atherosclerosis might also be interpreted in another way. If, in fact, atherosclerosis were a disease excessively affected by environmental factors such as dietary habits, it might be supposed that the nutritive habits would be carried on from generation to generation and so simulate a hereditary factor. However, in view of the change which diet in Sweden has undergone during the last 50 years, this interpretation is scarcely plausible.

Skeletal dimensions have been shown (VON VERSCHUER, 1954; LINDEGÅRD, 1954; and others) to be only very slightly affected by environment during adolescence

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and adult life. Consequently, the differences between type I and type II patients as regards length and sturdiness factors are highly suggestive of genotypic deviations in the two types of patient. Since, moreover, both a high length and high sturdiness factor were associated with the presence of hypercholesterolaemia in type I patients, the inference is that hypercholesterolaemia must be partly of genetic causation, no matter to what *additional* degree environment may have affected the serum cholesterol level.

A number of investigations have shown beyond doubt that the composition and size of the diet affects the serum cholesterol level and may be of atherogenic importance (see reviews by KEYS *et al*). Likewise, the nutritive environment is of importance to the accumulation of fat. However, the amount of body fat is also governed by genetic factors (LINDEGÅRD, MORSING and NYMAN, 1956).

In view of the results arrived at, one may conclude that an apparently healthy person showing the phenotypic features of our type I infarction patients should be managed prophylactically with dietary restrictions and possibly other measures to combat the manifestation of myocardial infarction. Although type I features comprise traits fairly uncommon in a group of normal persons such as our control series—hypercholesterolaemia and familial atherosclerosis—the combination of traits characteristic of this type is so conspicuous that it should readily be recognized in apparently healthy persons.

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Type II post-coronary patients were not characterized by a high serum cholesterol level or a high fat factor, but by a high degree of occupational leadership and probably by a higher degree of intellectual capacity and sub-solidity. These findings suggest that in this category of patient the onset is related more closely to the subject's mental environment than to his dietary habits. This agrees with the view that myocardial infarction is common in persons exposed to severe mental stress: the medical profession, officers, industrial leaders, high executives, and the like. The onset of myocardial infarction has also been reported to follow a relatively short period of mental stress (DUNBAR, 1948 and 1954).

Some of the phenotypic features of type II patients (high intellectual capacity and low solidity) may be taken to predispose to a high proportion of occupational leadership. Patients with high intellectual capacity and with personality traits corresponding to those termed sub-solidity in this paper may be presumed to be persons striving for prominent positions in the community. It should further be noted that the characteristics of type II were less consistent. This implies that a member of this category is more difficult to distinguish in a population of healthy persons. The results can scarcely allow any prediction to the effect that persons high in leadership and with other specified features are particularly exposed to the danger of myocardial infarction.

The recognition of two—at least—different basic types of post-coronary patient would appear to be of considerable importance to future research into preventative and therapeutic methods. We feel that advances in this sphere will profit by the distinction and deepened exploration of different types of post-coronary patient.

SUMMARY AND CONCLUSIONS

(1) The series studied comprised sixty-six male post-coronary patients aged between 40 and 55 years, selected from an urban population in the South of Sweden.

The following variables were studied: occupational, genealogic, anthropo-somatologic, psychologic, and biochemical—including determination of the serum cholesterol level and the urinary excretion of steroids. A random sample of controls, matched individually according to sex and age, was collected from the same population.

(2) Comparative studies of the post-coronary and control groups revealed certain differences, the patients tending towards *higher parental mortality due to atherosclerotic cardiovascular disease before the age of* 71, *higher incidence of obesity*, *higher incidence of periodic depressions*, *higher incidence of hypercholesterolaemia* ($\geqslant$300 mg/100 ml), and a *lower excretion of various steroid fractions in the urine* (17-ketosteroids, 17-ketosteroidogenic steroids, and tetrazoleum-reducing steroids, among others).

(3) An exhaustive search for relationships between various variables in patients and their matched controls, as well as in the members of the same group, suggested strongly that *the patient series was made up of different categories, or sub-groups, with distinctive combinations of the variables studied.* In this respect, some three-fourths of the patients could be assigned to either of two categories characterized by the following features:

Type I	*Type II*
Hypercholesterolaemia	Low or medium serum cholesterol level
High or medium excretion of the urinary steroids studied	Low excretion of the urinary steroids studied
History of parental atherosclerosis	High degree of occupational leadership
Explosive temperament	
Cyclothymous temperament (i.e. low degree of stability or high degree of viscerotonia);	Hysteroidous temperament (i.e. low degree of solidity according to SJÖBRING)
Large length and sturdiness factors of the skeleton	More than average intelligence (high capacity according to SJÖBRING)
Large muscle factor	Small length and sturdiness factors of the skeleton
Large fat factor	Light body-weight
Heavy body-weight	An interval more frequently exceeding 18 months between the onset of myocardial infarction and the examination

(4) The recognition of at least two distinct categories of post-coronary patient may afford some clues as to the symptomatology, future research on, and treatment of the postcoronary patient or candidate for the disease.

(a) Discrepancies in various series of undifferentiated postcoronary patients as compared with controls may be due to differences in composition of the series as wholes—the proportion of the sub-groups varying. In the present series, some of the features characterizing the patient series as a whole when compared with the controls could be ascribed chiefly to the sub-group termed type I (higher parental mortality from atherosclerotic disease, higher incidence of obesity, and higher incidence of hypercholesterolaemia). On the other hand, the lower mean excretion rate of the urinary steroids studied was mainly ascribable to the sub-group termed type II.

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It should be noted that some traits characterizing the sub-groups are masked when the patient series is treated statistically as a homogeneous whole (i.e. high body-weight, explosive temperament).

(b) The various features in the two sub-groups suggest more than one possible mechanism underlying coronary atherosclerosis and leading to myocardial infarction. Consequently, the recognition of different sub-groups may indicate different methods of research into the aetiology as well as the prevention and long-term treatment of the disease.

(5) The statistic relationship between different variables studied need not, of course, reflect a causal relationship. In some instances, however, the findings were believed to afford evidence of an aetiologic relationship. Thus, the hypercholersterolaemia of type I might include a genotypic component since it was associated with certain skeletal characteristics. Further, the high incidence of parental mortality due to atherosclerotic disease in type I bears out the assumption that hereditary factors are of importance in myocardial infarction—at least as regards the type I sub-group. Further multidisciplinary investigations are therefore called for.

(6) The present study was based on observations made on survivors of myocardial infarction. Had the patients who succumbed not been excluded, the findings of comparative studies between the patient and control groups might have been different. However, this selection had no influence on the main finding of the investigation—the heterogeneity of a group of patients with myocardial infarction.

(7) The features characterizing the sub-groups appear only little to have been influenced by factors connected with the onset of myocardial infarction and the subsequent convalescence. This is especially true of the skeletal dimensions and the occupational and genealogic variables. Consequently, the make-up of myocardial infarction candidates should be conspicuous among clinically 'healthy' persons. Particularly, the features characterizing type I patients are very distinctive and should permit of the institution of diagnostic and preventative measures.

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APPENDIX

This contains the following tables:

Table 4. Arithmetic mean values for the whole patient group and the whole control group.

Table 5. Comparison between all patients and their matched controls.

Tables 6(a) and 6(b). Comparison of the number of high, medium and low variants in all patients and their matched controls.

Table 7. Comparison between patients with (a) a high degree of leadership, (b) a medium or low degree of leadership, and their matched controls.

Tables 8(a) and 8(b). Comparison of the number of high, medium and low variants in patients with (a) high degree of leadership and (b) a medium or low degree of Leadership.

Table 9. Comparison between patients with (a) familial atherosclerosis, (b) without familial atherosclerosis, and their matched controls.

Tables 10(a) and 10(b). Comparison of the number of high, medium and low variants in patients with and without familial atherosclerosis.

Table 11. Comparison between patients with (a) a long, (b) a short survival time, and their matched controls.

Tables 12(a) and 12(b). Comparison of the number of high, medium and low variants in patients with a long and a short survival time.

Table 13. Comparison between patients with (a) a high length factor, (b) a medium or a low length factor, and their matched controls.

Tables 14(a) and 14(b). Comparison of the number of high, medium and low variants in patients and controls with (a) a high length factor and (b) a medium or low length factor.

Table 15. Comparison between patients with (a) a high sturdiness factor, (b) a medium or low sturdiness factor, and their matched controls.

Tables 16(a) and 16(b). Comparison of the number of high, medium and low variants in patients and controls with (a) a high sturdiness factor and (b) a medium or low sturdiness factor.

Table 17. Comparison between patients with (a) a high fat factor, (b) a medium or low fat factor, and their matched controls.

Tables 18(a) and 18(b). Comparison of the number of high, medium and low variants in patients and controls with (a) a high fat factor and (b) a medium or low fat factor.

Table 19. Comparison between patients with (a) high body-weight, (b) medium or low body-weight, and their matched controls.

Tables 20(a) and 20(b) Comparison of the number of high, medium and low variants in patients and controls with (a) high body-weight and (b) medium or low body-weight

Table 21. Comparison between patients (a) with, (b) without explosivity, and their matched controls.

Tables 22(a) and 22(b). Comparison of the number of high, medium and low variants in patients and controls with and without explosivity.

Table 23. Comparison between patients (a) with, (b) without periodic depressions, and their matched controls.

Tables 24(a) and 24(b). Comparison of the number of high, medium and low variants in patients with and without periodic depressions.

Table 25. Comparison between patients with (a) a high cholesterol value, (b) a medium or low cholesterol value, and their matched controls.

Tables 26(a) and 26(b). Comparison of the number of high, medium and low variants in patients with (a) a high cholesterol value and (b) a medium or low cholesterol value.

Table 27. Comparison between patients with (a) low urinary steroids, (b) medium or high urinary steroids, and their matched controls.

Tables 28(a) and 28(b). Comparison of the number of high, medium and low variants in patients and controls with (a) low urinary steroids and (b) medium or high urinary steroids.

Table 29. Comparison between patients (a) with, (b) without a history of peptic ulcer, and their matched controls.

Tables 30(a) and 30(b). Comparison of the number of high, medium and low variants in patients and controls with and without a history of peptic ulcer.

TABLE 4.—ARITHMETIC MEAN VALUES (M) FOR THE WHOLE PATIENT GROUP AND THE WHOLE CONTROL GROUP

	Patients			Controls		
	No.	M ± *εm*	S. D.	No.	M ± *εm*	S. D.
Tibial length (cm)	55	38·06 ± 0·24	1·80	55	38·37 ± 0·30	2·21
Radial length (cm)	55	25·75 ± 0·15	1·11	55	25·83 ± 0·15	1·11
Femoral condylar breadth (cm)	55	9·61 ± 0·06	0·42	55	9·65 ± 0·07	0·52
Bimalleolar width (cm)	55	7·23 ± 0·07	0·49	55	7·28 ± 0·05	0·37
Hand-grip strength (best hand)	45	38·82 ± 0·90	6·01	45	37·89 ± 0·73	4·87
Shoulder-thrust strength	41	41·20 ± 2·01	12·88	41	41·59 ± 1·85	11·83
Shoulder-pull strength	40	29·85 ± 0·92	5·82	40	32·08 ± 1·01	6·36
Body-weight (kg)	53	76·07 ± 1·36	9·91	53	75·06 ± 1·55	11·31
Body-length (cm)	53	171·80 ± 0·78	5·66	53	173·35 ± 0·86	6·25
Capacity	55	4·53 ± 0·15	1·12	55	4·29 ± 0·11	0·82
Validity	55	4·17 ± 0·15	1·13	55	4·19 ± 0·14	1·03
Stability	55	3·95 ± 0·16	1·19	55	4·01 ± 0·13	0·99
Solidity	55	3·53 ± 0·18	1·33	55	3·81 ± 0·13	0·94
17-KS (mg/24 hr)	60	10·28 ± 0·43	3·36	60	11·69 ± 0·50	3·87
17-KGS (mg/24 hr)	60	12·24 ± 0·46	3·53	60	13·01 ± 0·64	4·96

Skewly distributed values are not included

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TABLE 5.—COMPARISON BETWEEN ALL PATIENTS AND THEIR MATCHED CONTROLS

	All patients +	All patients −	Difference
Somatology			
Length factor			
Tibial length	24	31	
Radial length	23	29	
Mean value*	25	29	
Sturdiness factor			
Femoral condylar breadth	23	29	
Bimalleolar width	25	27	
Mean value*	24	27	
Muscle factor			
Hand-grip strength (best hand)	25	20	
Shoulder-thrust strength	19	20	
Shoulder-pull strength	14	22	
Mean value*	22	21	
Fat factor			
Skinfold subscapular	33	20	
Skinfold lateral thorax	34	21	
Skinfold abdomen	29	26	
Mean value*	33	22	
Body-weight	29	24	
Body-length	22	30	
Personality			
Capacity	20	15	
Validity	23	25	
Stability	21	26	
Solidity	21	25	
Serum cholesterol level	34	17	$0{\cdot}05 > p > 0{\cdot}01$
Urinary steroids			
17-KS	27	33	
17-KGS	24	35	
NF	23	34	
TZR	11	25	$0{\cdot}05 > p > 0{\cdot}01$
DHIA	25	29	
Mean value*	23	37	

* 'Mean value' is the term taken to express the average value for the part-variables of each of the somatic variables, sturdiness, muscle, and fat factors, as well as for the excretion of urinary steroids.

'Plus' and 'minus' here do not imply that all the part-variables showed higher or lower values than in the control, but that the majority of them did so.

\+ Denotes higher values for the control.

− Denotes lower values for the patient than for the control.

. No report of the degree of significance denotes a p-value greater than 0·05

TABLE 6(a).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN ALL PATIENTS AND THEIR MATCHED CONTROLS

		All patients	All controls	Difference
Occupational and family				
Leadership	+	14	7	
	M	17	21	
	—	31	34	
Physical activity	+	8	7	
	M	30	31	
	—	24	24	
Familial atherosclerosis	+	17	8	$0{\cdot}05 > p > 0{\cdot}01$
	—	45	54	
Somatology				
Length factor				
Tibial length	+	15	20	
	M	22	16	
	—	18	19	
Radial length	+	17	19	
	M	21	13	
	—	17	23	
Sturdiness factor				
Femoral condylar breadth	+	19	22	
	M	16	14	
	—	20	19	
Bimalleolar width	+	25	23	
	M	11	15	
	—	19	17	
Muscle factor				
Hand-grip strength (best hand)	+	19	12	
	M	10	18	
	—	16	15	
Shoulder-thrust strength	+	8	9	
	M	24	27	
	—	9	5	
Shoulder-pull strength	+	9	14	
	M	14	15	
	—	17	11	
Fat factor				
Skinfold subscapular	+	28	15	$p < 0{\cdot}01$
	M	15	16	
	—	12	24	
Skinfold lateral thorax	+	31	18	$0{\cdot}05 > p > 0{\cdot}01$
	M	12	19	
	—	12	18	
Skinfold abdomen	+	20	16	
	M	19	17	
	—	16	22	
Body-weight	+	22	17	
	M	19	17	
	—	12	19	
Body-length	+	12	18	
	M	25	19	
	—	16	16	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see pp. 100–102).
No report of the degree of significance denotes a p-value greater than 0·05.

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TABLE 6(b).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN ALL PATIENTS AND THEIR MATCHED CONTROLS

		All patients	All controls	Difference
Personality				
Capacity	+	29	27	
	M	19	21	
	—	7	7	
Validity	+	21	22	
	M	20	18	
	—	14	15	
Stability	+	18	21	
	M	16	17	
	—	21	17	
Solidity	+	10	16	
	M	17	13	
	—	28	26	
Explosivity	+	20	23	
	—	39	35	
Sensitivity to injustice	+	16	19	
	—	44	39	
Inability to forget suffered injustice	+	7	10	
	—	31	43	
Periodic depressions	+	12	3	$p < 0{\cdot}01$
	—	28	50	
Viscosity	+	14	16	
	—	24	37	
Impairment	+	5	4	
	—	54	54	
Neurotic traits	+	3	6	
	—	56	52	
Serum cholesterol level	+	17	6	
	M	32	35	$p < 0{\cdot}01$
	—	2	10	
Urinary steroids				
17-KS	+	14	17	
	M	20	23	
	—	26	20	
17-KGS	+	10	15	
	M	26	26	
	—	24	19	
NF	+	13	19	
	M	18	17	
	—	29	24	
TZR	+	10	17	
	M	16	14	
	—	11	6	
DHIA	+	10	19	
	M	14	13	
	—	36	28	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see pp. 100–102).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 7.—COMPARISON BETWEEN PATIENTS WITH (a) A HIGH DEGREE OF LEADERSHIP (b) A MEDIUM OR LOW DEGREE OF LEADERSHIP, AND THEIR MATCHED CONTROLS

	Patients				Difference
	Leaders v. matched controls		Non-leaders v. matched controls		
	+	−	+	−	
Somatology					
Length factor					
Tibial length	5	8	19	23	
Radial length	5	8	18	21	
Mean value*	4	7	16	19	
Sturdiness factor					
Femoral condylar breadth	4	8	19	21	
Bimalleolar width	5	6	20	21	
Mean value*	4	6	14	16	
Muscle factor					
Hand-grip strength (best hand)	5	5	20	15	
Shoulder-thrust strength	2	6	17	14	
Shoulder-pull strength	2	7	12	16	
Mean value*	4	6	18	16	
Fat factor					
Skinfold subscapular	7	6	26	14	
Skinfold lateral thorax	8	5	26	16	
Skinfold abdomen	8	5	20	21	
Mean value*	8	5	24	17	
Body-weight	5	7	25	17	
Body-length	6	7	16	23	
Personality					
Capacity	10	1	10	14	$p < 0{\cdot}01$
Validity	6	4	17	21	
Stability	7	3	14	23	
Solidity	3	6	18	19	
Serum cholesterol level	9	3	25	14	
Urinary steroids					
17-KS	4	9	23	24	
17-KGS	3	10	21	24	
NF	4	8	19	26	
TZR	2	6	9	19	
DHIA	5	7	20	22	
Mean value*	4	9	19	23	

* 'Mean value' is the term taken to express the average value for the part-variables of each of the somatic variables, sturdiness, muscle, and fat factors as well as for the excretion of urinary steroids.

'Plus' and 'minus' here do not imply that *all* the part-variables showed higher or lower values than in the control, but that the majority of them did so.

+ Denotes higher values for the patient than for the control.

− Denotes lower values for the patient than for the control.

No report of the degree of significance denotes a p-value greater than 0·05.

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TABLE 8(a).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS WITH (a) A HIGH DEGREE OF LEADERSHIP AND (b) A MEDIUM OR LOW DEGREE OF LEADERSHIP

		Patients		
		Leaders	Non-leaders	Difference
Occupational and family Physical activity	+	0	12	$p < 0{\cdot}01$
	M	2	28	
	—	12	8	
Familial atherosclerosis	+	5	12	
	—	9	32	
Somatology				
Length factor				
Tibial length	+	3	12	
	M	4	18	
	—	7	11	
Radial length	+	3	14	
	M	3	18	
	—	7	10	
Sturdiness factor				
Femoral condylar breadth	+	4	15	
	M	3	13	
	—	6	14	
Bimalleolar width	+	2	23	$p < 0{\cdot}01$
	M	2	9	
	—	9	10	
Muscle factor				
Hand-grip strength (best hand)	+	3	16	
	M	2	8	
	—	5	11	
Shoulder-thrust strength	+	1	7	
	M	5	19	
	—	2	7	
Shoulder-pull strength	+	1	8	
	M	7	7	
	—	1	16	
Fat factor				
Skinfold subscapular	+	7	21	
	M	3	12	
	—	3	9	
Skinfold lateral thorax	+	8	23	
	M	2	9	
	—	3	10	
Skinfold abdomen	+	4	16	
	M	4	15	
	—	5	11	
Body-weight	+	5	17	
	M	3	16	
	—	4	8	
Body-length	+	3	9	
	M	6	19	
	—	4	12	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 106).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 8(b).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS WITH (a) HIGH DEGREE OF LEADERSHIP AND (b) A MEDIUM OR LOW DEGREE OF LEADERSHIP

		Patients		Difference
		Leaders	Non-leaders	
Personality				
Capacity	+	12	17	
	M	1	18	$0{\cdot}05 > p > 0{\cdot}01$
	—	0	7	
Validity	+	8	13	
	M	3	17	$0{\cdot}05 > p > 0{\cdot}01$
	—	2	12	
Stability	+	5	13	
	M	3	13	
	—	5	16	
Solidity	+	1	9	
	M	3	14	
	—	9	19	
Explosivity	+	3	18	
	—	10	27	
Sensitivity to injustice	+	2	10	
	—	6	18	
Inability to forget suffered injustice	+	1	6	
	—	7	22	
Periodic depressions	+	3	8	
	—	5	20	
Viscosity	+	4	9	
	—	4	19	
Impairment	+	0	5	
	—	13	40	
Neurotic traits	+	0	2	
	—	13	43	
Serum cholesterol level	+	5	12	
	M	6	26	
	—	1	1	
Urinary steroids				
17-KS	+	1	13	
	M	8	12	
	—	4	22	
17-KGS	+	1	9	
	M	7	19	
	—	5	19	
NF	+	1	12	
	M	5	13	
	—	7	22	
TZR	+	3	8	
	M	3	12	
	—	2	9	
DHIA	+	1	9	
	M	3	11	
	—	9	27	

+ Denotes high values.
M Denotes medium values.
— Denotes low values. (For calculations, see p. 106).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 9.—COMPARISON BETWEEN PATIENTS (a) WITH FAMILIAL ATHEROSCLEROSIS AND (b) WITHOUT FAMILIAL ATHEROSCLEROSIS AND THEIR MATCHED CONTROLS

	Patients				Difference
	Patients with fam. ath. v. matched controls		Patients without fam. ath. v. matched controls		
	+	−	+	−	
Somatology					
Length factor					
Tibial length	7	9	17	22	
Radial length	6	7	17	22	
Mean value*	8	7	17	22	
Sturdiness factor					
Femoral condylar breadth	6	9	17	20	
Bimalleolar width	9	7	16	20	
Mean value*	7	8	17	19	
Muscle factor					
Hand-grip strength (best hand)	6	6	19	14	
Shoulder-thrust strength	6	5	13	15	
Shoulder-pull strength	5	3	9	19	
Mean value*	7	4	15	17	
Fat factor					
Skinfold subscapular	10	4	23	16	
Skinfold lateral thorax	12	4	22	17	
Skinfold abdomen	9	7	20	19	
Mean value*	11	5	22	17	
Personality					
Body-weight	9	5	20	19	
Body-length	4	10	18	20	
Capacity	8	3	12	12	
Validity	4	10	19	15	
Stability	2	9	19	17	$0{\cdot}05 < p < 0{\cdot}01$
Solidity	7	7	14	18	
Serum cholesterol level	9	6	25	11	
Urinary steroids					
17-KS	9	8	18	25	
17-KGS	7	9	17	26	
NF	10	6	13	28	$0{\cdot}05 < p < 0{\cdot}01$
TZR	2	7	9	18	
DHIA	9	5	16	24	
Mean value*	9	8	14	29	

* 'Mean value' is the term taken to express the average value for the part-variables of each of the somatic variables, sturdiness, muscle, and fat factors, as well as for the excretion of urinary steroids.

'Plus' and 'minus' here do not imply that *all* the part-variables showed higher or lower values than in the control, but that the majority of them did so.

\+ Denotes higher values for the patient than for the control.

− Denotes lower values for the patient than for the control.

No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 10(a).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS WITH AND WITHOUT FAMILIAL ATHEROSCLEROSIS

		Patients		Difference
		Familial atherosclerosis		
		Present	Absent	
Occupational and family				
Leadership	+	5	9	
	M	5	12	
	—	7	20	
Physical activity	+	1	6	
	M	7	20	
	—	9	15	
Somatology				
Length factor				
Tibial length	+	2	13	
	M	8	14	
	—	6	12	
Radial length	+	2	15	
	M	9	12	
	—	5	12	
Sturdiness factor				
Femoral condylar breadth	+	4	15	
	M	5	11	
	—	7	13	
Bimalleolar width	+	6	19	
	M	2	9	
	—	8	11	
Muscle factor				
Hand-grip strength (best hand)	+	3	16	
	M	3	7	
	—	6	10	
Shoulder-thrust strength	+	2	6	
	M	7	17	
	—	7	7	
Shoulder-pull strength	+	2	7	
	M	6	8	
	—	3	14	
Fat factor				
Skinfold subscapular	+	7	21	
	M	7	8	
	—	2	10	
Skinfold lateral thorax	+	9	22	
	M	5	7	
	—	2	10	
Skinfold abdomen	+	8	12	
	M	4	15	
	—	4	12	
Body-weight	+	7	15	
	M	5	14	
	—	2	10	
Body-length	+	1	11	
	M	9	16	
	—	5	11	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 106).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 10(b).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS WITH AND WITHOUT FAMILIAL ATHEROSCLEROSIS

		Patients		Difference
		Familial atherosclerosis		
		Present	Absent	
Personality				
Capacity	+	12	17	$0{\cdot}05 > p > 0{\cdot}01$
	M	4	15	
	—	0	7	
Validity	+	5	16	
	M	6	14	
	—	5	9	
Stability	+	4	14	
	M	4	12	
	—	8	13	
Solidity	+	2	8	
	M	7	10	
	—	7	21	
Explosivity	+	9	11	
	—	8	31	
Sensitivity to injustice	+	5	11	
	—	12	32	
Inability to forget suffered injustice	+	1	6	
	—	11	20	
Periodic depressions	+	5	7	
	—	7	19	
Viscosity	+	5	9	
	—	7	17	
Impairment	+	1	4	
	—	16	38	
Neurotic traits	+	0	3	
	—	17	39	
Serum cholesterol level	+	4	13	
	M	10	22	
	—	1	1	
Urinary steroids				
17-KS	+	2	12	
	M	9	11	
	—	6	20	
17-KGS	+	3	7	
	M	9	17	
	—	5	19	
NF	+	3	10	
	M	5	13	
	—	9	20	
TZR	+	3	7	
	M	2	17	
	—	4	7	
DHIA	+	3	7	
	M	2	12	
	—	12	24	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 106).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 11.—COMPARISON BETWEEN PATIENTS WITH (a) A LONG (b) A SHORT SURVIVAL TIME, AND THEIR MATCHED CONTROLS

	Patients				Difference
	Time between examination and onset of disease				
	≤18 months v. matched controls		>18 months v. matched controls		
	+	−	+	−	
Somatology					
Length factor					
Tibial length	9	14	15	17	
Radial length	10	11	13	18	
Mean value*	9	10	11	16	
Sturdiness factor					
Femoral condylar breadth	14	7	9	22	$p < 0{\cdot}01$
Bimalleolar width	11	10	14	17	
Mean value*	11	7	7	15	
Muscle factor					
Hand-grip strength (best hand)	13	4	12	16	$0{\cdot}05 > p > 0{\cdot}01$
Shoulder-thrust strength	8	5	11	15	
Shoulder-pull strength	5	8	9	15	
Mean value*	10	6	11	16	
Fat factor					
Skinfold subscapular	14	8	19	12	
Skinfold lateral thorax	14	9	20	12	
Skinfold abdomen	14	9	15	17	
Mean value*	13	9	19	13	
Body-weight	16	6	13	18	$0{\cdot}05 > p > 0{\cdot}01$
Body-length	8	13	14	17	
Personality					
Capacity	7	3	13	12	
Validity	10	10	13	15	
Stability	9	12	12	14	
Solidity	10	10	11	15	
Serum cholesterol level	14	7	18	9	
Urinary steroids					
17-KS	11	15	15	18	
17-KGS	7	19	16	16	
NF	9	17	13	17	
TZR	5	16	6	9	
DHIA	10	14	15	15	
Mean value*	8	16	14	17	

* 'Mean value' is the term taken to express the average value for the part-variables of each of the somatic variables, sturdiness, muscle, and fat factors, as well as for the excretion of urinary steroids.

'Plus' and 'minus' here do not imply that *all* the part-variables showed higher or lower values than in the control, but that the majority of them did so.

+ Denotes higher values for the patient than for the control.

− Denotes lower values for the patient than for the control.

No report of the degree of significance denotes a p-value greater than 0·05.

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TABLE 12(a).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS WITH A LONG AND A SHORT SURVIVAL TIME

		Patients		Difference
		Time between examination and onset of disease		
		≤18 months	>18 months	
Occupational and family Leadership	+	8	6	
	M	7	10	
	—	13	18	
Physical activity	+	4	4	
	M	13	17	
	—	11	13	
Familial atherosclerosis	+	9	8	
	—	18	23	
Somatology Length factor Tibial length	+	5	10	
	M	10	12	
	—	8	10	
Radial length	+	4	13	
	M	14	7	
	—	5	12	
Sturdiness factor Femoral condylar breadth	+	11	8	
	M	7	9	$0{\cdot}05 > p > 0{\cdot}01$
	—	5	15	
Bimalleolar width	+	11	14	
	M	5	6	
	—	7	12	
Muscle factor Hand-grip strength (best hand)	+	9	10	
	M	4	6	
	—	4	12	
Shoulder-thrust strength	+	4	4	
	M	8	16	
	—	3	6	
Shoulder-pull strength	+	4	5	
	M	6	8	
	—	4	13	
Fat factor Skinfold subscapular	+	13	15	
	M	7	8	
	—	3	9	
Skinfold lateral thorax	+	15	17	
	M	6	6	
	—	3	9	
Skinfold abdomen	+	9	11	
	M	8	11	
	—	6	10	
Body-weight	+	12	11	
	M	9	11	
	—	4	9	
Body-length	+	5	7	
	M	11	14	
	—	6	10	

+ Denotes high values. M Denotes medium values. — Denotes low values.
No report of the degree of significance denotes a p-value greater than 0·05.

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TABLE 12(b).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS WITH A LONG AND A SHORT SURVIVAL TIME

		Patients		Difference
		Time between examination and onset of disease		
		≤18 months	>18 months	
Personality				
Capacity	+	13	16	
	M	9	10	
	—	1	6	
Validity	+	10	11	
	M	11	9	$0{\cdot}05 > p > 0{\cdot}01$
	—	2	12	
Stability	+	7	11	
	M	7	9	
	—	9	12	
Solidity	+	3	7	
	M	7	10	
	—	13	15	
Explosivity	+	11	10	
	—	15	22	
Sensivity to injustice	+	6	6	
	—	13	11	
Inability to forget suffered injustice	+	4	3	
	—	15	14	
Periodic depressions	+	3	8	
	—	16	9	$0{\cdot}05 > p > 0{\cdot}01$
Viscosity	+	7	6	
	—	12	11	
Impairment	+	4	1	
	—	22	31	
Neurotic traits	+	0	2	
	—	26	30	
Serum cholesterol level	+	6	11	
	M	13	18	
	—	2	0	
Urinary steroids				
17-KS	+	6	8	
	M	8	11	
	—	12	14	
17-KGS	+	4	5	
	M	12	14	
	—	10	14	
NF	+	7	6	
	M	7	11	
	—	12	16	
TZR	+	6	4	
	M	8	8	
	—	5	4	
DHIA	+	5	5	
	M	6	8	
	—	15	20	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 107).
No report of the degree of significance denotes a p-value greater than 0·05.

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TABLE 13.—COMPARISON BETWEEN PATIENTS WITH (a) A HIGH LENGTH FACTOR AND (b) A MEDIUM OR A LOW LENGTH FACTOR AND THEIR MATCHED CONTROLS

	Patients with high length factor v. matched controls		Patients with med. or low length factor v. matched controls		Difference
	+	−	+	−	
Somatology					
Sturdiness factor					
Femoral condylar breadth	10	10	13	19	
Bimalleolar width	12	8	13	19	
Mean value*	8	6	10	16	
Muscle factor					
Hand-grip strength (best hand)	10	7	15	13	
Shoulder-thrust strength	6	8	13	12	
Shoulder-pull strength	6	8	8	15	
Mean value*	8	8	15	12	
Fat factor					
Skinfold subscapular	12	7	21	13	
Skinfold lateral thorax	13	7	21	14	
Skinfold abdomen	11	10	18	16	
Mean value*	12	7	20	15	
Body-weight	13	7	16	17	
Body-length	12	8	10	22	$0{\cdot}05 > p > 0{\cdot}01$
Personality					
Capacity	9	5	11	10	
Validity	9	8	14	17	
Stability	11	7	10	19	$0{\cdot}05 > p > 0{\cdot}01$
Solidity	10	8	11	17	$p < 0{\cdot}01$
Serum cholesterol level	15	3	19	13	
Urinary steroids					
17-KS	10	10	16	23	
17-KGS	10	10	13	25	
NF	7	12	14	25	
TZR	2	6	9	19	
DHIA	8	10	17	18	
Mean value*	8	9	13	24	

* 'Mean value' is the term taken to express the average value for the part-variables of each of the somatic variables, sturdiness, muscle, and fat factors, as well as for the excretion of urinary steroids.

'Plus' and 'minus' here do not imply that *all* the part-variables showed higher or lower values than in the control, but that the majority of them did so.

+ Denotes higher values for the patient than for the control.

− Denotes lower values for the patient than for the control.

No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 14(a).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH (a) A HIGH LENGTH FACTOR AND (b) A MEDIUM OR LOW LENGTH FACTOR

		Patients			Controls		
		Length factor		Difference	Length factor		Difference
		High	Med. or low		High	Med. or low	
Occupational and family							
Leadership	+	3	11		4	5	
	M	9	8		12	18	
	—	10	21		16	31	
Physical activity	+	4	4		2	7	
	M	10	20		15	30	
	—	8	16		15	17	
Familial atherosclerosis	+	3	14				
	—	19	22				
Somatology							
Sturdiness factor							
Femoral condylar breadth	+	11	10		16	15	
	M	6	11		8	13	
	—	5	16		8	21	
Bimalleolar width	+	13	15		18	14	
	M	3	8		10	19	$p < 0{\cdot}01$
	—	6	14		4	16	
Muscle factor							
Hand-grip strength (best hand)	+	8	11		16	12	$0{\cdot}05 > p > 0{\cdot}01$
	M	4	8		{8	{22	
	—	6	11		{6	{13	
Shoulder-thrust strength	+	1	7		11	11	
	M	11	16		16	30	
	—	4	5		2	4	
Shoulder-pull strength	+	4	5		14	18	
	M	3	12		10	15	
	—	8	10		5	12	
Fat factor							
Skinfold subscapular	+	11	19		7	16	
	M	5	11		10	11	
	—	6	7		15	22	
Skinfold lateral thorax	+	15	18		9	16	
	M	1	11		12	18	
	—	6	8		12	15	
Skinfold abdomen	+	9	12		7	11	
	M	6	15		11	20	
	—	7	10		14	18	
Body-weight	+	13	12		13	12	
	M	7	14		11	17	
	—	4	10		8	20	
Body-length	+	12	2		22	2	
	M	9	17	$p < 0{\cdot}01$	9	20	$p < 0{\cdot}01$
	—	1	16		1	26	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 108).
No report of the degree of significance denotes a p-value greater than 0·05.

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TABLE 14(b).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH (a) A HIGH LENGTH FACTOR AND (b) A MEDIUM OR LOW LENGTH FACTOR

		Patients		Difference	Controls		Difference
		Length factor			Length factor		
		High	Med. or low		High	Med. or low	
Personality							
Capacity	+	13	20		13	17	
	M	7	13		14	24	
	—	4	5		5	8	
Validity	+	8	14		15	16	
	M	8	15		9	17	
	—	8	9		8	16	
Stability	+	11	11		16	13	
	M	8	9	+	8	17	
	—	5	18		8	19	
Solidity	+	7	4		8	17	
	M	6	11		9	15	
	—	11	23		15	17	
Explosivity	+	8	13		13	22	
	—	14	23		19	27	
Sensitivity to injustice	+	3	9		11	18	
	—	9	15		16	26	
Inability to forget suffered injustice	+	3	4		5	10	
	—	9	20		22	35	
Periodic depressions	+	2	9		0	5	
	—	10	15		27	40	
Viscosity	+	2	11		7	12	
	—	10	13		20	33	
Impairment	+	1	4		1	5	
	—	21	32		31	44	
Neurotic traits	+	1	1		4	7	
	—	21	35		28	42	
Serum cholesterol level	+	10	8		0	8	
	M	9	25	$0{\cdot}05 > p > 0{\cdot}01$	20	33	$0{\cdot}05 > p > 0{\cdot}01$
	—	1	1		4	7	
Urinary steroids							
17-KS	+	4	11		14	15	
	M	10	11		11	18	
	—	8	19		7	18	
17-KGS	+	4	6		10	10	
	M	7	20		14	26	
	—	11	15		8	15	
NF	+	4	11		13	21	
	M	8	10		10	15	
	—	10	20		9	14	
TZR	+	3	9		8	14	
	M	3	13		9	13	
	—	4	9		2	7	
DHIA	+	2	9		12	17	
	M	9	6		6	12	
	—	11	26		14	22	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 108).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 15.—COMPARISON BETWEEN PATIENTS WITH (a) A HIGH STURDINESS FACTOR, (b) A MEDIUM OR LOW STURDINESS FACTOR, AND THEIR MATCHED CONTROLS

	Patients with high sturdiness factor v. matched controls		Patients with med. or low sturdiness factor v. matched controls		Difference
	+	−	+	−	
Somatology					
Length factor					
Tibial length	12	12	12	19	
Radial length	13	10	10	19	
Mean value*	11	9	9	17	
Muscle factor					
Hand-grip strength (best hand)	13	6	12	14	
Shoulder-thrust strength	11	6	8	14	
Shoulder-pull strength	6	10	8	13	
Mean value*	12	7	10	15	
Fat factor					
Skinfold subscapular	15	8	18	12	
Skinfold lateral thorax	15	9	19	12	
Skinfold abdomen	12	12	17	14	
Mean value*	13	10	19	12	
Body-weight	18	6	11	18	$p < 0{\cdot}01$
Body-length	12	10	10	20	
Personality					
Capacity	6	9	14	6	
Validity	10	10	13	15	
Stability	8	13	13	13	
Solidity	12	11	9	14	
Serum cholesterol level	14	6	20	10	
Urinary steroids					
17-KS	14	11	12	22	
17-KGS	10	15	13	20	
NF	11	13	11	21	
TZR	4	11	7	14	
DHIA	11	13	14	16	
Mean value*	12	12	10	21	

* 'Mean value' is the term taken to express the average value for the part-variables of each of the somatic variables, sturdiness, muscle, and fat factors, as well as for the excretion of urinary steroids.

'Plus' and 'minus' here do not imply that *all* the part-variables showed higher or lower values than in the control, but that the majority of them did so.

+ Denotes higher values for the patient than for the control.

− Denotes lower values for the patient than for the control.

No report of the degree of significance denotes a p-value greater than 0·05.

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TABLE 16(a).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH (a) A HIGH STURDINESS FACTOR AND (b) A MEDIUM OR LOW STURDINESS FACTOR

		Patients			Controls		
		Sturdiness factor		Difference	Sturdiness factor		Difference
		High	Med. or low		High	Med. or low	
Occupational and family Leadership	+	3	11		5	4	
	M	7	10		15	15	
	—	16	15		14	33	
Physical activity	+	4	3		2	7	
	M	13	17		18	27	
	—	9	16		14	18	
Familial atherosclerosis	+	6	11				
	—	19	22				
Somatology Length factor Tibial length	+	11	8		19	12	
	M	11	12		6	13	$0{\cdot}05 > p > 0{\cdot}01$
	—	7	13		9	22	
Radial length	+	10	10		14	10	
	M	12	10		9	15	
	—	7	13		11	21	
Muscle factor Hand-grip strength (best hand)	+	11	10		16	12	
	M	9	5	$0{\cdot}05 > p > 0{\cdot}01$	7	23	
	—	3	15		9	10	
Shoulder-thrust strength	+	3	6		13	9	
	M	16	15		16	30	
	—	3	8		2	4	
Shoulder-pull strength	+	6	4		20	12	
	M	6	11		8	17	$p < 0{\cdot}01$
	—	8	12		3	14	
Fat factor Skinfold subscapular	+	14	16		10	13	
	M	11	7		11	10	
	—	4	10		13	24	
Skinfold lateral thorax	+	16	19		12	12	
	M	8	5		16	14	$p < 0{\cdot}01$
	—	5	9		6	21	
Skinfold abdomen	+	10	13		8	10	
	M	15	7		20	11	
	—	4	13		6	26	
Body-weight	+	16	9		19	6	
	M	11	10	$p < 0{\cdot}01$	11	17	$p < 0{\cdot}01$
	—	2	12		4	24	
Body-length	+	11	5		18	6	
	M	10	16		9	20	$p < 0{\cdot}01$
	—	8	11		6	21	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 108).
No report of the degree of significance denotes a p-value greater than 0·05

TABLE 16(b).—COMPARISON OF THE NUMBER OF HIGH MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH (a) A HIGH STURDINESS FACTOR AND (b) A MEDIUM OR LOW STURDINESS FACTOR

		Patients		Difference	Controls		Difference
		Sturdiness factor			Sturdiness factor		
		High	Med. or low		High	Med. or low	
Personality							
Capacity	+	13	21		9	21	
	M	11	9		18	20	
	—	5	3		6	7	
Validity	+	10	12		17	14	
	M	12	11		11	15	$0{\cdot}05 > p > 0{\cdot}01$
	—	7	10		5	19	
Stability	+	11	11		10	19	
	M	7	10		9	16	
	—	11	12		14	13	
Solidity	+	6	5		11	14	
	M	7	10		9	15	
	—	16	18		13	19	
Explosivity	+	12	9		15	20	
	—	14	23		18	28	
Sensitivity to injustice	+	8	4		10	19	
	—	9	15		19	22	
Inability to forget suffered injustice	+	5	3		6	9	
	—	12	16		24	33	
Periodic depressions	+	5	6		2	3	
	—	12	13		28	39	
Viscosity	+	6	7		10	9	
	—	11	12		20	33	
Impairment	+	2	3		3	3	
	—	24	29		30	45	
Neurotic traits	+	1	1		3	8	
	—	25	31		30	40	
Serum cholesterol level	+	10	10		2	6	
	M	16	23		21	32	
	—	0	2		5	6	
Urinary steroids							
17-KS	+	9	6		15	14	
	M	9	12		9	20	
	—	11	18		8	17	
17-KGS	+	3	7		9	11	
	M	11	16		14	26	
	—	15	13		9	14	
NF	+	11	4		12	22	
	M	5	13		11	14	
	—	13	19		9	15	
TZR	+	6	7		11	11	
	M	7	9		5	17	
	—	6	7		3	6	
DHIA	+	6	5		12	17	
	M	9	6		6	12	
	—	14	25		14	22	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 108).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 17.—COMPARISON BETWEEN PATIENTS WITH (a) A HIGH FAT FACTOR, (b) A MEDIUM OR LOW FAT FACTOR AND THEIR MATCHED CONTROLS

	Patients with high fat factor v. matched controls		Patients with med. or low fat factor v. matched controls		Difference
	+	−	+	−	
Somatology					
Length factor					
Tibial length	11	17	13	14	
Radial length	12	15	11	14	
Mean value*	10	15	10	12	
Sturdiness factor					
Femoral condylar breadth	12	15	11	14	
Bimalleolar width	13	13	12	14	
Mean value*	9	10	9	12	
Muscle factor					
Hand-grip strength (best hand)	13	9	12	10	
Shoulder-thrust strength	8	11	10	8	
Shoulder-pull strength	5	11	9	11	
Mean value*	11	11	11	8	
Body-weight	19	8	10	16	$0{\cdot}05 > p > 0{\cdot}01$
Body-length	12	14	10	16	
Personality					
Capacity	13	6	7	9	
Validity	16	9	7	16	$0{\cdot}05 > p > 0{\cdot}01$
Stability	9	16	11	10	
Solidity	9	14	12	11	
Serum cholesterol level	19	5	15	12	$p = 0{\cdot}05$
Urinary steroids					
17-KS	12	16	14	16	
17-KGS	10	18	13	17	
NF	10	17	12	17	
TZR	4	11	7	14	
DHIA	12	14	13	15	
Mean value*	10	15	12	18	

* 'Mean value' is the term taken to express the average value for the part-variables of each of the somatic variables, sturdiness, muscle, and fat factors, as well as for the excretion of urinary steroids.

'Plus' and 'minus' here do not imply that *all* the part-variables showed higher or lower values than in the control, but that the majority of them did so.

\+ Denotes higher values for the patient than for the control.

− Denotes lower values for the patient than for the control.

No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 18(a).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH (a) A HIGH FAT FACTOR AND (b) A MEDIUM OR LOW FAT FACTOR

		Patients Fat factor		Difference	Controls Fat factor		Difference
		High	Med. or low		High	Med. or low	
Occupational and family							
Leadership	+	8	6		4	5	
	M	9	8		9	21	
	—	11	20		9	38	
Physical activity	+	2	6		2	7	
	M	12	18		11	35	
	—	14	10		9	22	
Familial atherosclerosis	+	8	9				
	—	19	22				
Somatology							
Length factor							
Tibial length	+	8	7		10	21	
	M	10	12		6	13	
	—	10	8		6	25	
Radial length	+	9	8		6	18	
	M	12	9		7	18	
	—	7	10		9	23	
Sturdiness factor							
Femoral condylar breadth	+	10	9		9	22	
	M	11	5		8	14	
	—	7	13		5	23	
Bimalleolar width	+	10	15		9	23	
	M	6	5		6	23	
	—	12	7		7	13	
Muscle factor							
Hand-grip strength (best hand)	+	8	11		9	19	
	M	5	5		8	22	
	—	10	6		4	15	
Shoulder-thrust strength	+	4	4		6	16	
	M	9	15		13	33	
	—	7	2		2	4	
Shoulder-pull strength	+	4	5		10	22	
	M	5	9		8	17	
	—	10	7		3	14	
Body-weight	+	15	8		13	12	
	M	9	11	$0{\cdot}05 > p > 0{\cdot}01$	9	19	$p < 0{\cdot}01$
	—	3	10		0	28	
Body-length	+	5	6		8	16	
	M	14	12		5	24	
	—	7	10		8	19	

+ Denotes high values. M Denotes medium values. — Denotes low values. (For calculations, see p. 109). No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 18(b).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH (a) A HIGH FAT FACTOR AND (b) A MEDIUM OR LOW FAT FACTOR

		Patients Fat factor		Difference	Controls Fat factor		Difference
		High	Med. or low		High	Med. or low	
Personality							
Capacity	+	17	12		5	25	
	M	9	10		12	25	
	—	2	5		5	8	
Validity	+	14	6		17	13	
	M	6	14	$0{\cdot}05 > p > 0{\cdot}01$	5	21	$p < 0{\cdot}01$
	—	7	7		0	24	
Stability	+	8	10		6	23	
	M	6	10		4	21	$0{\cdot}05 > p > 0{\cdot}01$
	—	14	7		12	14	
Solidity	+	5	5		4	21	
	M	8	9		7	17	
	—	15	13		11	20	
Explosivity	+	11	10		8	27	
	—	17	20		14	32	
Sensitivity to injustice	+	5	7		6	23	
	—	11	13		12	27	
Inability to forget suffered injustice	+	2	5		3	12	
	—	14	15		15	43	
Periodic depressions	+	6	5		1	4	
	—	10	15		17	51	
Viscosity	+	7	6		4	15	
	—	9	14		14	40	
Impairment	+	1	4		0	6	
	—	27	26		22	53	
Neurotic traits	+	0	2		3	9	
	—	28	28		19	50	
Serum cholesterol level	+	11	9		2	6	
	M	13	26		12	41	
	—	2	0		4	7	
Urinary steroids							
17-KS	+	6	8		6	23	
	M	11	8		9	20	
	—	11	15		7	18	
17-KGS	+	5	4		5	15	
	M	12	14		12	28	
	—	11	13		5	18	
NF	+	5	8		7	27	
	M	10	8		7	18	
	—	13	15		8	16	
TZR	+	4	6		5	17	
	M	7	9		4	18	
	—	4	7		2	7	
DHIA	+	3	7		6	15	
	M	9	5		5	8	
	—	16	19		11	18	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 109).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 19.—COMPARISON BETWEEN PATIENTS WITH (a) HIGH BODY-WEIGHT, (b) MEDIUM OR LOW BODY-WEIGHT AND THEIR MATCHED CONTROLS

	Patients with high body-weight v. matched controls		Patients with med. or low body-weight v. matched controls		Difference
	+	−	+	−	
Somatology					
Length factor					
Tibial length	12	10	12	21	
Radial length	11	10	12	19	
Mean value*	11	9	9	17	
Sturdiness factor					
Femoral condylar breadth	15	7	7	22	$p < 0{\cdot}01$
Bimalleolar width	12	9	13	18	
Mean value*	11	5	6	17	$p < 0{\cdot}01$
Muscle factor					
Hand-grip strength (best hand)	10	8	15	12	
Shoulder-thrust strength	8	9	11	11	
Shoulder-pull strength	6	10	8	13	
Mean value*	8	10	14	12	
Fat factor					
Skinfold subscapular	15	6	18	14	
Skinfold lateral thorax	18	4	16	17	$0{\cdot}05 > p > 0{\cdot}01$
Skinfold abdomen	14	8	15	18	
Mean value*	16	5	16	17	$0{\cdot}05 > p > 0{\cdot}01$
Body-length	12	10	10	20	
Personality					
Capacity	9	6	11	9	
Validity	11	9	12	16	
Stability	5	14	16	12	$0{\cdot}05 > p > 0{\cdot}01$
Solidity	11	9	10	16	
Serum cholesterol level	16	3	18	13	$p = 0{\cdot}05$
Urinary steroids					
17-KS	13	9	12	24	
17-KGS	15	8	8	27	$p < 0{\cdot}01$
NF	10	11	12	23	
TZR	4	11	6	14	
DHIA	12	10	13	19	
Mean value*	13	8	9	25	$p < 0{\cdot}01$

* 'Mean value' is the term taken to express the average value for the part-variables of each of the somatic variables, sturdiness, muscle, and fat factors, as well as for the excretion of urinary steroids.

'Plus' and 'minus' here do not imply that *all* the part-variables showed higher or lower values than in the control, but that the majority of them did so.

\+ Denotes higher values for the patient than for the control.

− Denotes lower values for the patient than for the control.

No report of the degree of significance denotes a p-value greater than 0·05.

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TABLE 20(a).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH (a) HIGH BODY-WEIGHT AND (b) MEDIUM OR LOW BODY-WEIGHT

		Patients body-weight		Difference	Controls body-weight		Difference
		High	Med. or low		High	Med. or low	
Occupational and family Leadership	+	5	9		4	5	
	M	7	10		11	19	
	—	11	20		10	37	
Physical activity	+	5	3		1	8	
	M	8	22		11	34	
	—	10	14		13	19	
Familial atherosclerosis	+	7	10				
	—	15	26				
Somatology Length factor Tibial length	+	7	8		13	18	
	M	11	11		6	13	
	—	4	14		6	25	
Radial length	+	9	8		11	13	
	M	9	12		8	17	$0{\cdot}05 > p > 0{\cdot}01$
	—	4	13		6	26	
Sturdiness factor Femoral condylar breadth	+	13	6		17	14	
	M	6	10	$p < 0{\cdot}01$	7	14	$p < 0{\cdot}01$
	—	3	17		1	28	
Bimalleolar width	+	13	12		17	15	
	M	3	8		5	24	$p < 0{\cdot}01$
	—	6	13		3	17	
Muscle factor Hand-grip strength (best hand)	+	8	11		12	16	
	M	6	4		7	23	
	—	4	12		5	14	
Shoulder-thrust strength	+	2	6		11	11	
	M	14	13		12	34	
	—	4	5		1	5	
Shoulder-pull strength	+	5	4		18	14	
	M	5	9		6	19	$p < 0{\cdot}01$
	—	7	10		0	17	
Fat factor Skinfold subscapular	+	14	14		13	10	
	M	8	7		8	13	$p < 0{\cdot}01$
	—	0	12		4	33	
Skinfold lateral thorax	+	16	15		15	9	
	M	5	7	$0{\cdot}05 < p < 0{\cdot}01$	9	21	$p < 0{\cdot}01$
	—	1	11		1	26	
Skinfold abdomen	+	8	12		11	7	
	M	11	8		13	18	$p < 0{\cdot}01$
	—	3	13		1	31	
Body-length	+	7	5		14	10	
	M	13	12	$0{\cdot}05 < p < 0{\cdot}01$	6	23	$p < 0{\cdot}01$
	—	2	14		5	22	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 109).

No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 20(b).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH (a) HIGH BODY-WEIGHT AND (b) MEDIUM OR LOW BODY-WEIGHT

		Patients body-weight		Difference	Controls body-weight		Difference
		High	Med. or low		High	Med. or low	
Personality							
Capacity	+	13	16		8	22	
	M	4	15		13	24	
	—	5	2		4	9	
Validity	+	12	9		14	16	
	M	5	15	$0{\cdot}05 > p > 0{\cdot}01$	9	17	$p < 0{\cdot}01$
	—	5	9		2	22	
Stability	+	4	14		7	22	
	M	5	11	$0{\cdot}05 > p > 0{\cdot}01$	7	18	
	—	13	8		11	15	
Solidity	+	8	2		7	18	
	M	4	13	$0{\cdot}05 > p > 0{\cdot}01$	7	17	
	—	10	18		11	20	
Explosivity	+	13	8	$0{\cdot}05 > p > 0{\cdot}01$	12	23	
	—	10	27		13	33	
Sensitivity to injustice	+	8	4		6	23	
	—	9	15		17	25	
Inability to forget suffered injustice	+	5	2		2	13	
	—	12	17		21	37	
Periodic depressions	+	7	4		2	3	
	—	10	15		21	47	
Viscosity	+	8	5		5	14	
	—	9	14		18	36	
Impairment	+	0	5	$0{\cdot}05 > p > 0{\cdot}01$	1	5	
	—	23	30		24	51	
Neurotic traits	+	1	1		4	8	
	—	22	34		21	48	
Serum cholesterol level	+	9	11		1	7	
	M	10	26		18	35	
	—	1	1		4	7	
Urinary steroids							
17-KS	+	8	6		9	20	
	M	7	12		9	20	
	—	8	18		7	18	
17-KGS	+	6	3		8	12	
	M	9	17		8	32	
	—	8	16		9	14	
NF	+	5	8		8	26	
	M	8	10		8	17	
	—	10	18		9	15	
TZR	+	6	4		7	15	
	M	6	10		5	17	
	—	3	8		2	7	
DHIA	+	4	6		9	20	
	M	6	8		7	11	
	—	13	22		9	27	

+ Denotes high values. M Denotes medium values. — Denotes low values.

(For calculations, see p. 109).

No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 21.—COMPARISON BETWEEN PATIENTS (a) WITH AND (b) WITHOUT EXPLOSIVITY AND THEIR MATCHED CONTROLS

	Patients with explosivity v. matched controls		Patients without explosivity v. matched controls		Difference
	+	−	+	−	
Somatology					
Length factor					
Tibial length	8	11	16	20	
Radial length	8	9	15	20	
Mean value*	8	10	12	16	
Sturdiness factor					
Femoral condylar breadth	11	8	11	22	
Bimalleolar width	11	8	14	19	
Mean value*	9	5	9	17	
Muscle factor					
Hand-grip strength (best hand)	9	5	15	16	
Shoulder-thrust strength	10	4	9	17	$0{\cdot}05 > p > 0{\cdot}01$
Shoulder-pull strength	4	7	10	17	
Mean value*	9	5	13	17	
Fat factor					
Skinfold subscapular	12	6	21	14	
Skinfold lateral thorax	15	4	19	17	
Skinfold abdomen	11	8	18	18	
Mean value*	12	6	20	16	
Body-weight	13	5	15	20	$0{\cdot}05 > p > 0{\cdot}01$
Body-length	7	12	15	18	
Personality					
Capacity	5	6	15	9	
Validity	7	11	16	14	
Stability	2	12	19	14	$p < 0{\cdot}01$
Solidity	9	8	12	17	
Serum cholesterol level	10	6	24	11	
Urinary steroids					
17-KS	10	10	17	23	
17-KGS	8	12	16	23	
NF	12	7	11	27	$0{\cdot}05 > p > 0{\cdot}01$
TZR	5	9	6	16	
DHIA	11	7	14	22	
Mean value*	9	8	13	25	

* 'Mean value' is the term taken to express the average value for the part-variables of each of the somatic variables, sturdiness, muscle, and fat factors, as well as for the excretion of urinary steroids.

'Plus' and 'minus' here do not imply that *all* the part-variables showed higher or lower values than in the control, but that the majority of them did so.

\+ Denotes higher values for the patient than for the control.

− Denotes lower values for the patient than for the control.

No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 22(a).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH AND WITHOUT EXPLOSIVITY

		Patients explosivity		Difference	Controls explosivity		Difference
		Present	Absent		Present	Absent	
Occupational and family Leadership	+	3	11		4	5	
	M	4	13		12	18	
	—	13	18		19	28	
Physical activity	+	4	4		3	6	
	M	8	22		16	29	
	—	8	16		16	16	
Familial atherosclerosis	+	9	8	$0{\cdot}05 > p > 0{\cdot}01$			
	—	10	31				
Somatology Length factor Tibial length	+	5	10		11	17	
	M	7	15		10	11	
	—	7	11		11	17	
Radial length	+	5	12		9	14	
	M	10	11		12	12	
	—	4	13		12	18	
Sturdiness factor Femoral condylar breadth	+	8	11		13	15	
	M	6	10		8	12	
	—	5	15		11	18	
Bimalleolar width	+	11	14		11	19	
	M	1	10		13	15	
	—	7	12		8	12	
Muscle factor Hand-grip strength (best hand)	+	5	13		11	10	
	M	3	7		9	15	
	—	4	12		7	12	
Shoulder-thrust strength	+	2	5		7	8	
	M	8	16		16	21	
	—	2	7		2	4	
Shoulder-pull strength	+	2	7		9	13	
	M	4	10		12	10	
	—	4	12		5	9	
Fat factor Skinfold subscapular	+	11	17		9	13	
	M	6	9		9	11	
	—	2	10		15	20	
Skinfold lateral thorax	+	14	17		8	14	
	M	2	10		12	15	
	—	3	9		12	15	
Skinfold abdomen	+	9	11		6	12	
	M	6	14		14	13	
	—	4	11		13	19	
Body-weight	+	12	10		9	14	
	M	5	14	$p < 0{\cdot}01$	12	14	
	—	1	11		11	15	
Body-length	+	5	5		9	14	
	M	9	15		11	17	
	—	4	8		12	8	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 111).
No report of the degree of significance denotes a p-value greater than 0·05.

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TABLE 22(b).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH AND WITHOUT EXPLOSIVITY

		Patients explosivity		Difference	Controls explosivity		Difference
		Present	Absent		Present	Absent	
Personality							
Capacity	+	7	22		14	19	
	M	8	11		13	18	
	—	4	3		6	7	
Validity	+	7	14		12	17	
	M	7	13		13	11	
	—	5	9		8	16	
Stability	+	3	15		14	15	
	M	4	12	$p < 0{\cdot}01$	7	16	
	—	12	9		12	13	
Solidity	+	4	6		9	15	
	M	5	12		8	14	
	—	10	18		16	15	
Sensitivity to injustice	+	10	2	$p < 0{\cdot}01$	19	10	$p < 0{\cdot}01$
	—	7	17		13	29	
Inability to forget suffered injustice	+	5	2		7	8	
	—	12	17		25	31	
Periodic depressions	+	7	4		3	2	
	—	10	15		29	37	
Viscosity	+	9	4		10	9	
	—	8	15		22	30	
Impairment	+	0	5		3	3	
	—	21	31		32	43	
Neurotic traits	+	0	2		3	8	
	—	21	34		32	38	
Serum cholesterol level	+	6	11		6	2	
	M	9	23		22	28	
	—	1	1		4	7	
Urinary steroids							
17-KS	+	5	9		9	20	
	M	8	12		12	17	
	—	7	19		12	14	
17-KGS	+	5	5		11	9	
	M	6	20		12	28	
	—	9	15		10	14	
NF	+	6	7		12	22	
	M	4	14		8	17	
	—	10	19		13	12	
TZR	+	5	5		8	13	
	M	4	12		6	13	
	—	5	6		1	7	
DHIA	+	5	4		13	16	
	M	3	11		4	14	
	—	11	25		16	21	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 111).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 23.—COMPARISON BETWEEN PATIENTS (a) WITH, (b) WITHOUT PERIODIC DEPRESSIONS, AND THEIR MATCHED CONTROLS

	Patients with periodic depressions v. matched controls		Patients without periodic depressions v. matched controls		Difference
	+	−	+	−	
Somatology					
Length factor					
Tibial length	4	7	20	24	
Radial length	5	6	18	23	
Mean value*	3	5	17	21	
Sturdiness factor					
Femoral condylar breadth	6	5	17	24	
Bimalleolar width	6	4	19	23	
Mean value*	4	2	14	20	
Muscle factor					
Hand-grip strength (best hand)	3	6	22	14	
Shoulder-thrust strength	4	5	15	15	
Shoulder-pull strength	2	5	12	18	
Mean value*	4	5	18	17	
Fat factor					
Skinfold subscapular	7	3	26	17	
Skinfold lateral thorax	7	4	27	17	
Skinfold abdomen	6	5	23	21	
Mean value*	6	4	26	18	
Body-weight	7	4	22	20	
Body-length	4	7	18	23	
Personality					
Capacity	4	2	16	13	
Validity	3	7	20	18	
Stability	1	7	20	18	$0{\cdot}05 > p > 0{\cdot}01$
Solidity	4	5	17	20	
Serum cholesterol level	6	4	28	13	
Urinary steroids					
17-KS	7	4	20	29	
17-KGS	5	5	19	30	
NF	4	6	19	28	
TZR	2	6	9	19	
DHIA	5	6	20	23	
Mean value*	4	6	19	27	

* 'Mean value' is the term taken to express the average value for the part-variables of each of the somatic variables, sturdiness, muscle, and fat factors, as well as for the excretion of urinary steroids.

'Plus' and 'minus' here do not imply that *all* the part-variables showed higher or lower values than in the control, but that the majority of them did so.

+ Denotes higher values for the patient than for the control.

− Denotes lower values for the patient than for the control.

No report of the degree of significance denotes a p-value greater than 0·05.

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TABLE 24(a).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS WITH AND WITHOUT PERIODIC DEPRESSIONS

		Patients periodic depressions		Difference
		Present	Absent	
Occupational and family Leadership	+	3	11	
	M	3	14	
	—	5	26	
Physical activity	+	1	7	
	M	5	25	
	—	5	19	
Familial atherosclerosis	+	5	12	
	—	6	35	
Somatology Length factor Tibial length	+	1	14	
	M	8	14	
	—	2	16	
Radial length	+	2	15	
	M	5	16	
	—	4	13	
Sturdiness factor Femoral condylar breadth	+	4	15	
	M	4	12	
	—	3	17	
Bimalleolar width	+	5	20	
	M	3	8	
	—	3	16	
Muscle factor Hand-grip strength (best hand)	+	3	16	
	M	2	8	
	—	4	12	
Shoulder-thrust strength	+	2	6	
	M	4	20	
	—	3	6	
Shoulder-pull strength	+	3	6	
	M	2	12	
	—	2	15	
Fat factor Skinfold subscapular	+	6	22	
	M	2	13	
	—	3	9	
Skinfold lateral thorax	+	6	25	
	M	1	11	
	—	4	8	
Skinfold abdomen	+	4	16	
	M	4	15	
	—	3	13	
Body-weight	+	7	15	
	M	1	18	
	—	3	9	
Body-length	+	2	10	
	M	7	18	
	—	2	14	

+ Denotes high values. M Denotes medium values. — Denotes low values.

(For calculations, see p. 112).

No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 24(b).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS WITH AND WITHOUT PERIODIC DEPRESSIONS

		Patients periodic depressions		Difference
		Present	Absent	
Personality				
Capacity	+	7	22	
	M	2	17	
	—	2	5	
Validity	+	4	17	
	M	1	19	
	—	6	8	
Stability	+	1	17	
	M	3	13	$0{\cdot}05 > p > 0{\cdot}01$
	—	7	14	
Solidity	+	2	8	
	M	3	14	
	—	6	22	
Explosivity	+	7	10	
	—	4	14	
Sensitivity to injustice	+	7	5	
	—	4	19	$p < 0{\cdot}01$
Inability to forget suffered injustice	+	3	4	
	—	8	20	
Viscosity	+	5	8	
	—	6	16	
Impairment	+	0	4	
	—	11	20	
Neurotic traits	+	1	1	
	—	10	23	
Serum cholesterol level	+	4	13	
	M	6	26	
	—	0	2	
Urinary steroids				
17-KS	+	2	12	
	M	5	15	
	—	4	22	
17-KGS	+	2	8	
	M	5	21	
	—	4	20	
NF	+	2	11	
	M	3	15	
	—	6	23	
TZR	+	3	7	
	M	4	12	
	—	1	10	
DHIA	+	2	8	
	M	0	14	
	—	9	27	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 112).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 25.—COMPARISON BETWEEN PATIENTS WITH (a) A HIGH CHOLESTEROL VALUE, (b) A MEDIUM OR LOW CHOLESTEROL VALUE, AND THEIR MATCHED CONTROLS

	Patients with high serum cholesterol v. matched controls		Patients with med. or low serum cholesterol v. matched controls		Difference
	+	−	+	−	
Somatology					
Length factor					
Tibial length	10	6	11	18	
Radial length	8	5	11	18	
Mean value*	10	6	8	15	
Sturdiness factor	9	7	8	18	
Bimalleolar width	10	6	10	18	
Mean value*	8	4	7	16	$0{\cdot}05 > p > 0{\cdot}01$
Muscle factor					
Hand-grip strength (best hand)	10	4	10	12	
Shoulder-thrust strength	5	6	7	11	
Shoulder-pull strength	3	8	8	10	
Mean value*	8	6	9	12	
Fat factor					
Skinfold subscapular	10	6	17	10	
Skinfold lateral thorax	11	5	15	13	
Skinfold abdomen	10	6	14	15	
Mean value*	10	7	15	12	
Body-weight	13	2	11	17	$p < 0{\cdot}01$
Body-length	9	7	10	18	
Personality					
Capacity	2	8	14	5	$p < 0{\cdot}01$
Validity	9	5	10	15	
Stability	4	11	13	10	
Solidity	7	8	10	12	
Urinary steroids					
17-KS	11	5	9	21	$0{\cdot}05 > p > 0{\cdot}01$
17-KGS	9	7	10	20	
NF	8	7	11	19	
TZR	0	6	5	16	
DHIA	8	6	10	19	
Mean value*	9	4	7	23	$p < 0{\cdot}01$

* 'Mean value' is the term taken to express the average value for the part-variables of each of the somatic variables, sturdiness, muscle, and fat factors, as well as for the excretion of urinary steroids.

'Plus' and 'minus' here do not imply that *all* the part-variables showed higher or lower values than in the control, but that the majority of them did so.

\+ Denotes higher values for the patient than for the control.

− Denotes lower values for the patient than for the control.

No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 26(a).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS WITH (a) A HIGH CHOLESTEROL VALUE AND (b) A MEDIUM OR LOW CHOLESTEROL VALUE

		Patients		Difference
		Serum cholesterol		
		High	Med. or low	
Occupational and family				
Leadership	+	5	9	
	M	2	15	
	—	10	21	
Physical activity	+	3	5	
	M	8	22	
	—	6	18	
Family atherosclerosis	+	4	13	
	—	12	29	
Somatology				
Length factor				
Tibial length	+	8	6	
	M	5	12	
	—	3	11	
Radial length	+	7	8	
	M	5	11	
	—	4	10	
Sturdiness factor				
Femoral condylar breadth	+	9	7	
	M	3	9	
	—	4	13	
Bimalleolar width	+	9	10	
	M	3	6	
	—	4	13	
Muscle factor				
Hand-grip strength (best hand)	+	6	9	
	M	4	4	
	—	4	9	
Shoulder-thrust strength	+	2	3	
	M	6	14	
	—	4	3	
Shoulder-pull strength	+	2	6	
	M	4	7	
	—	7	6	
Fat factor				
Skinfold subscapular	+	9	13	
	M	5	9	
	—	2	7	
Skinfold lateral thorax	+	12	14	
	M	2	8	
	—	2	7	
Skinfold abdomen	+	7	9	$0{\cdot}05 > p > 0{\cdot}01$
	M	8	10	
	—	1	10	
Body-weight	+	9	10	
	M	5	10	
	—	1	8	
Body-length	+	6	4	
	M	7	15	
	—	3	10	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 112).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 26(b).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS WITH (a) A HIGH CHOLESTEROL VALUE AND (b) A MEDIUM OR LOW CHOLESTEROL VALUE

		Patients		Difference
		Serum cholesterol		
		High	Med. or low	
Personality				
Capacity	+	6	17	
	M	6	11	$0{\cdot}05 > p > 0{\cdot}01$
	—	4	1	
Validity	+	8	9	
	M	4	13	
	—	4	7	
Stability	+	5	10	
	M	4	10	
	—	7	9	
Solidity	+	4	5	
	M	6	8	
	—	6	16	
Explosivity	+	7	14	
	—	9	28	
Sensitivity to injustice	+	4	8	
	—	4	20	
Inability to forget suffered injustice	+	3	4	
	—	5	24	
Periodic depressions	+	4	7	
	—	4	21	
Viscosity	+	3	10	
	—	5	18	
Impairment	+	0	5	
	—	16	37	
Neurotic traits	+	1	1	
	—	15	41	
Urinary steroids				
17-KS	+	4	6	$p > 0{\cdot}01$
	M	9	7	
	—	3	17	
17-KGS	+	3	4	
	M	6	16	
	—	7	11	
NF	+	2	7	
	M	9	6	
	—	5	18	
TZR	+	0	4	
	M	4	9	
	—	2	8	
DHIA	+	2	6	
	M	5	3	
	—	8	22	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 112).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 27.—COMPARISON BETWEEN PATIENTS WITH (a) LOW URINARY STEROIDS, (b) MEDIUM OR HIGH URINARY STEROIDS, AND THEIR MATCHED CONTROLS

	Patients with low urinary steroids v. matched controls		Patients with med. or high urinary steroids v. matched controls		Difference
	+	−	+	−	
Somatology					
Length factor					
Tibial length	9	15	15	16	
Radial length	10	12	13	17	
Mean value*	8	11	11	15	
Sturdiness factor					
Femoral condylar breadth	11	11	12	18	
Bimalleolar width	12	11	13	16	
Mean value*	9	9	8	13	
Muscle factor					
Hand-grip strength (best hand)	14	7	11	13	
Shoulder-thrust strength	10	8	9	10	
Shoulder-pull strength	8	8	6	15	
Mean value*	11	8	11	13	
Fat factor					
Skinfold subscapular	16	6	17	14	
Skinfold lateral thorax	17	5	19	16	
Skinfold abdomen	14	10	15	16	
Mean value*	16	7	16	15	
Body-weight	13	10	16	14	
Body-length	6	15	16	14	
Personality					
Capacity	10	5	10	10	
Validity	10	10	13	15	
Stability	9	13	12	13	
Solidity	11	10	10	15	
Serum cholesterol level	14	9	20	7	

* 'Mean value' is the term taken to express the average value for the part-variables of each of the somatic variables, sturdiness, muscle, and fat factors, as well as for the excretion of urinary steroids.

'Plus' and 'minus' here do not imply that *all* the part-variables showed higher or lower values than in the control, but that the majority of them did so.

\+ Denotes higher values for the patient than for the control.

− Denotes lower values for the patient than for the control.

No report of the degree of significance denotes a p-value greater than 0·05.

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TABLE 28(a).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH (a) LOW URINARY STEROIDS AND (b) MEDIUM OR HIGH URINARY STEROIDS

		Patients urinary steroids		Difference	Controls urinary steroids		Difference
		Low	Med. or high		Low	Med. or high	
Occupational and family Leadership	+	4	10		1	8	
	M	10	7		4	26	$0{\cdot}05 > p > 0{\cdot}01$
	—	13	18		16	30	
Physical activity	+	5	3		4	4	
	M	12	18		12	33	
	—	10	14		5	27	
Familial atherosclerosis	+	7	11				
	—	21	23				
Somatology Length factor Tibial length	+	5	10		7	23	
	M	10	12		6	13	
	—	9	9		6	25	
Radial length	+	7	10		5	19	
	M	8	13		5	20	
	—	9	8		9	23	
Sturdiness factor Femoral condylar breadth	+	8	11		8	23	
	M	6	10		4	17	
	—	10	10		7	22	
Bimalleolar width	+	12	13		8	24	
	M	4	7		5	24	
	—	8	11		6	14	
Muscle factor Hand-grip strength (best hand)	+	10	10		6	22	
	M	5	5		6	24	
	—	6	10		6	13	
Shoulder-thrust strength	+	1	7		6	16	
	M	15	9		9	37	
	—	3	6		3	3	
Shoulder-pull strength	+	4	5		8	24	
	M	6	7		4	21	
	—	7	10		6	11	
Fat factor Skinfold subscapular	+	11	17		6	17	
	M	8	7		5	16	
	—	5	7		8	29	
Skinfold lateral thorax	+	14	17		6	18	
	M	6	6		7	23	
	—	4	8		6	21	
Skinfold abdomen	+	11	9		4	14	
	M	6	13		8	23	
	—	7	9		7	25	
Body-weight	+	9	14		7	18	
	M	9	11		7	19	
	—	6	7		5	23	
Body-length	+	2	10		5	19	
	M	12	13	$0{\cdot}05 > p > 0{\cdot}01$	7	22	
	—	9	7		7	20	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 114).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 28(b).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH (a) LOW URINARY STEROIDS AND (b) MEDIUM OR HIGH URINARY STEROIDS

		Patients urinary steroids		Difference	Controls urinary steroids		Difference
		Low	Med. or high		Low	Med. or high	
Personality							
Capacity	+	15	14		6	24	
	M	7	12		10	27	
	—	2	5		3	10	
Validity	+	8	13		6	24	
	M	9	11		11	15	
	—	7	7		2	22	
Stability	+	6	12		7	22	
	M	6	9		4	21	
	—	11	10		8	18	
Solidity	+	4	6		6	19	
	M	10	7		3	21	
	—	10	18		10	21	
Explosivity	+	9	13		9	26	
	—	18	22		11	35	
Sensitivity to injustice	+	5	9		5	24	
	—	13	13		11	28	
Inability to forget suffered injustice	+	1	7		2	13	
	—	17	15		14	44	
Periodic depressions	+	5	7		0	5	
	—	13	15		16	52	
Viscosity	+	3	12	$0{\cdot}05 > p > 0{\cdot}01$	3	16	
	—	15	10		13	41	
Impairment	+	2	4		3	3	
	—	25	31		17	58	
Neurotic traits	+	1	2		2	10	
	—	26	33		18	51	
Serum cholesterol level	+	4	13		2	6	
	M	19	12	$0{\cdot}05 > p > 0{\cdot}01$	13	40	
	—	0	2		2	9	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 114).
No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 29.—COMPARISON BETWEEN PATIENTS (a) WITH, (b) WITHOUT A HISTORY OF PEPTIC ULCER, AND THEIR MATCHED CONTROLS

	Patients with ulcer v. matched controls		Patients without ulcer v. matched controls		Difference
	+	−	+	−	
Somatology					
Length factor					
Tibial length	6	4	18	27	
Radial length	5	4	18	25	
Mean value*	5	3	15	23	
Sturdiness factor					
Femoral condyler breadth	4	6	19	23	
Bimalleolar width	4	4	21	23	
Mean value*	4	5	14	18	
Muscle factor					
Hand-grip strength (best hand)	4	2	21	18	
Shoulder-thrust strength	2	3	16	17	
Shoulder-pull strength	2	4	12	19	
Mean value*	2	3	20	19	
Fat factor					
Skinfold subscapular	7	3	26	17	
Skinfold lateral thorax	5	5	29	16	
Skinfold abdomen	5	5	24	21	
Mean value*	6	4	26	18	
Body-weight	3	6	24	19	
Body-length	5	5	18	25	
Personality					
Capacity	1	4	19	11	
Validity	3	5	20	20	
Stability	4	5	17	21	
Solidity	2	5	19	20	
Serum cholesterol level	8	2	26	15	
Urinary steroids					
17-KS	4	6	22	27	
17-KGS	3	7	21	28	
NF	3	7	20	27	
TZR	2	5	9	20	
DHIA	2	5	23	24	
Mean value*	2	7	21	26	

* 'Mean value' is the term taken to express the average value for the part-variables of each of the somatic variables, sturdiness, muscle, and fat factors, as well as for the excretion of urinary steroids.

'Plus' and 'minus' here do not imply that *all* the part-variables showed higher or lower values than in the control, but that the majority of them did so.

+ Denotes higher values for the patient than for the control.

— Denotes lower values for the patient than for the control.

No report of the degree of significance denotes a p-value greater than 0·05.

TABLE 30(a).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH AND WITHOUT A HISTORY OF PEPTIC ULCER

		Patients		Difference	Controls		Difference
		Ulcer individuals	Others		Ulcer individuals	Others	
Occupational and family							
Leadership	+	2	12		1	6	
	M	1	16		3	17	
	—	8	23		7	28	
Physical activity	+	1)	7)	$0{\cdot}05 > p > 0{\cdot}01$	1	6	
	M	9)	21)		6	25	
	—	1	23		4	20	
Familial atherosclerosis	+	2	15				
	—	7	34				
Somatology							
Length factor							
Tibial length	+	3	14		2	18	
	M	3	19		4	12	
	—	4	15		5	14	
Radial length	+	3	16		3	16	
	M	4	17		2	11	
	—	3	15		6	17	
Sturdiness factor							
Femoral condylar breadth	+	2	18		4	18	
	M	4	14		3	11	
	—	4	16		4	15	
Bimalleolar width	+	4	22		4	19	
	M	3	10		6	9	
	—	3	16		1	16	
Muscle factor							
Hand-grip strength (best hand)	+	3	18		2	10	
	M	1	10		4	14	
	—	3	14		4	11	
Shoulder-thrust strength	+	1	8		2	7	
	M	4	24		7	20	
	—	2	9		0	5	
Shoulder-pull strength	+	0	10		3	11	
	M	3	13		3	12	
	—	4	14		2	9	
Fat factor							
Skinfold subscapular	+	3	25		2	14	
	M	4	11		3	14	
	—	3	9		6	19	
Skinfold lateral thorax	+	6	25		3	16	
	M	1	11		2	19	
	—	3	9		6	12	
Skinfold abdomen	+	4	16		1	15	
	M	3	16		3	17	
	—	3	13		7	15	
Body-weight	+	1	22		3	14	
	M	5	15	$0{\cdot}05 > p > 0{\cdot}01$	2	15	
	—	4	9		6	13	
Body-length	+	2	12		1	17	
	M	4	21		7	12	
	—	4	14		3	13	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 114).
No report of the degree of significance denotes a p-value greater than 0·05.

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TABLE 30(b).—COMPARISON OF THE NUMBER OF HIGH, MEDIUM AND LOW VARIANTS IN PATIENTS AND CONTROLS WITH AND WITHOUT A HISTORY OF PEPTIC ULCER

		Patients		Difference	Controls		Difference
		Ulcer individuals	Others		Ulcer individuals	Others	
Personality							
Capacity	+	2	29		6	21	
	M	6	13		4	17	
	—	2	6		1	6	
Validity	+	3	18		4	18	
	M	4	17		4	14	
	—	3	13		3	12	
Stability	+	1	19		6	15	
	M	5	11		3	14	
	—	4	18		2	15	
Solidity	+	0	11		1	15	
	M	4	13		2	11	(+)
	—	6	24		8	18	
Explosivity	+	3	18		5	29	
	—	7	30		9	38	
Sensitivity to injustice	+	3	9		5	24	
	—	6	18		6	35	
Inability to forget suffered injustice	+	2	5		3	12	
	—	7	22		8	49	
Periodic depressions	+	2	8		1	4	
	—	7	16		10	57	
Viscosity	+	4	9		5	14	
	—	5	18		6	47	
Impairment	+	1	4		2	4	
	—	9	44		12	63	
Neurotic traits	+	0	2		1	11	
	—	10	46		13	56	
Serum cholesterol level	+	4	15		2	5	
	M	6	31		6	30	
	—	0	2		3	7	
Urinary steroids							
17-KS	+	0	14		3	14	
	M	4	16		1	22	
	—	7	20		6	14	
17-KGS	+	2	8		1	14	
	M	5	21		4	22	
	—	4	21		5	14	
NF	+	1	12		3	17	
	M	4	14		3	18	
	—	6	25		4	15	
TZR	+	1	10		2	15	
	M	6	10		2	13	
	—	2	10		1	5	
DHIA	+	1	6		4	15	
	M	1	11		1	12	
	—	9	19		5	23	

+ Denotes high values. M Denotes medium values. — Denotes low values.
(For calculations, see p. 114).
No report of the degree of significance denotes a p-value greater than 0·05.

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THE APPLICATION OF LEARNING THEORY TO THE TREATMENT OF STAMMERING

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IF we regard psychiatric symptoms as products of learning processes, obeying certain laws as laid down principally by HULL (1943), then it will become apparent why modern learning theory together with subsequent theoretical refinement, can offer much in its therapeutic application to the treatment of psychiatrically-ill patients (JONES, 1956; MOWRER, 1950; RAYMOND, 1956; WALTON and BLACK; YATES.).

The present paper is an attempt to derive from learning theory a method of eliminating an abnormal reaction, on the part of a patient, which had failed to respond to other therapeutic means; to apply this method in an individual case; to relate the procedure adopted to a theoretical rationale; and finally to enquire into the possible limitations of this theoretical model.

SUMMARY OF THE CASE HISTORY

The patient, a young man of 32 years, was admitted to hospital for the investigation and treatment of a chronic stammer. In ordinary conversation his stammer was slight, but when he was forced to use the telephone it became much more severe.

Stammering first occurred at the age of eight or nine. The cause was unknown. It was less acute at home and his parents did not apparently appreciate its full effects. At school however he was ashamed of his affliction, feeling it was regarded as a weakness. To counteract this impression he indulged in acts of extreme bravado and daredevilry. To avoid stammering in class he pretended not to know the aswers to questions put to him. Ambition and intelligence, however, repeatedly led him into situations where the stammer would be noticed, and at grammer school no good was to be served in not answering questions if he was to get on and rise above the poor background from which he had come. He discovered numerous 'dodges' whereby stammering was minimized, for instance for most stammer-provoking words there were alternatives with the same meaning. But as school work became more technical, requiring use of specific terms, he abandoned ideas of trying for college and left school to take up an electrical apprenticeship.

In the army he continued his trade and also matriculated. The fact that promotion eluded him he attributes to the stammer. Despite further indications of his academic potentiality evidenced in his army career he nevertheless still saw his stammer as too big a handicap and on leaving the army became a draughtsman. He studied electrical engineering at night school and rapidly obtained his H.N.C. By now he was in the design office of the Atomic Energy Authority surrounded by colleagues who were college trained and whom he noticed were no more competent than himself. Again ambition for a post of leadership and responsibility seemed realizable but for the need to keep his stammer hidden. The telephone, an integral part of an engineer's working life, now became the stumbling block. Ability to speak clearly was all it called for but this he could not do. As a youth using the 'phone in private, speaking to someone he knew well, the stammer was controllable. But now, in a large office, full of people, speaking to complete strangers, on topics requiring specific terminology, without the assistance of personal appearance, facial expression or gesture, his stammer was revealed in its worst form. Although he knew he was ideally qualified for promotion the prospect of increased use of the telephone prevented him applying for promotion and he watched less able men by-pass him. With his wife and family, as with his parents, the stammer was minimized, but

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in the telephone situation it was aggravated and he had been running away from his stammer too long to face up to it now.

Although there appeared to be a satisfactory rationale and theoretical explanation (next section) of his inability to use the telephone without stammering it was possible that factors other than those of stimulus generalization were creating or exacerbating his difficulties. The most obvious of these was that he might be ill-equiped to cope with the increasing intellectual demands of his job or that he did not possess the other abilities relevant to his position.

The General Aptitude Test Battery (1947), the Wechsler-Bellevue Intelligence Scale, Form 1 (1944) and the Kuder Vocational Interest Test (1951) results were not significantly different from those returned by people successful in engineering and designing occupations.

Both the Minnesota (1951) and the Maudsley Personality Inventories (1957) returned abnormal scores. The high scores on the hypomania and extraversion scales would be consistent with the intense drive state mentioned previously, whilst the high "neuroticism" score might be in accordance with his early history of a low stress threshold.

All in all the psychological test results are not inconsistent with the development of his stammer in terms of the theoretical model proposed below.

SUGGESTED DEVELOPMENT OF THE STAMMER IN TERMS OF LEARNING THEORY

There is experimental evidence to suggest that the stronger the habit strength, the closer must be the excitatory stimuli to the original stimulus aggregate. It follows that a considerable number of stimuli are able in varying degrees to evoke the same reaction. This spreading of learning to other stimuli has been called by HULL 'primary stimulus generalization'.

Those circumstances which precipitated the stammer in its acute form, and therefore presumably approaching most closely the original stimulus aggregate were those in which he was required to provide an answer in class or to answer a roll call. The common denominators of these appeared to be the presence of other people and the necessity to make an independent contribution.

The stammering could, therefore, be regarded as a drive-reducing conditioned avoidance response, evoked originally in a traumatic situation. In such a situation intense anxiety would have been generated and the evocation of stammering would have resulted in a reduction or cessation of the anxiety-inducing stimulus. The reaction potential of the stammer would have developed as the product of the habit strength and the fluctuating drive strength (anxiety). The habit strength of the stammer would have increased as a simple negatively accelerated positive growth function, eventually reaching an asymptote or saturation point beyond which similar excitatory stimuli would not have increased the habit strength.

If the stammer is regarded as a conditioned avoidance response arising from the anxiety and the anxiety as a conditioned response to a stress situation, then in similar stress situations anxiety should develop. The patient whose case we are here considering reports that he avoided the direct use of his voice in many situations, e.g. at a tube station by buying tickets from the machines, and when taking a girl to the cinema by having bought the tickets earlier in the day. Thus the anxiety which would have normally developed in such situations gradually dissipated, and since the anxiety resulted in stammering, the stammering would also diminish. Similarly, in other situations, by suppressing the conditioned avoidance response with alcohol, or capitalizing on his personal appearance, a reduction of anxiety would again be produced and again in turn cause a decline in stammering. This pattern, reproduced over the years, is given further emphasis by another feature, namely, his failure to

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stammer when in the company of his wife and family. Here the effect of stimulus generalization as described earlier would appear important in that his wife and family had little in common with the original stimulus aggregate; anxiety was, therefore, not developed; and since the stammer is a conditioned avoidance response arising from the anxiety, the stammer tends not to occur.

In addition, stimuli which become associated with the drive state assume secondary motivating values, whilst stimuli associated with the reinforcement of drive reduction acquire secondary reinforcing values. In other words there develops a conditioned drive for success and praise, both of these values acquiring secondary motivating characteristics. At the same time the stimuli in which these secondary motivating characteristics occur would acquire secondary reinforcing values. Through a process of discrimination these secondary mechanisms would either be retained or extinguished according to their effect. Since presumably this patient's stress threshold is lower than normal, a greater zone of stimuli would be extinguished than retained and he would be left with a continually reinforced drive state limited to a few selected conditions.

Thus the conditioned drive for success and praise forced him into those situations which, because of stimulus generalization, were the most traumatic and anxiety-inducing. The use of the telephone was one of these.

SYMPTOMATIC TREATMENT OF THE STAMMERING

(a) *Theoretical considerations*

HULL's fundamental formula linking performance, habit and drive is:

$$S^{E}R = S^{H}R \times D$$

where performance is symbolized by $S^{E}R$ and habit by $S^{H}R$. In other words performance is a multiplicative function of both habit and drive. If there is no drive there is no performance, although the habit may be a strong one. If the drive remains constant the suggested approach would be to develop a negative habit, a "habit of not responding to the stimulus". The reaction tendency of stammering should then decrease as a negative growth function of the frequency of occurrence of closely associated and successively unreinforced evocations, i.e. the number of times the telephone is used and stammering does not occur.

The work of CHERRY *et al.* (1953, 1955, 1956) on the treatment of stammering provided a method of minimizing the stammering whilst using the telephone. Their work indicated that stammering was a perceptual rather than a motor abnormality and, as such, amenable to training and conditioning. Their method was to interfere with the speakers' auditory perceptions of his own voice. Their initial experiments used a "speech shadowing" technique, a means whereby part of the speaker's auditory perception is taken away from his own voice and transferred to that of another speaker. The experimenter reads from a book and the patient shadows him, without seeing the text, following one or two words behind.

The development of the habit strength, i.e. of not responding to the telephone with a stammer would proceed as a function of the following seven considerations:

(i) *The number of reinforcements.* Repetition is of itself of little value in strengthening habits but merely provides the opportunity for learning. The main variable is reinforcement.

(ii) *The importance of the reinforcing agent* (i.e. of not stammering).

(iii) *The interval between S–R.* There should be an immediate reward with no delay between the *S–R*.

(iv) *Massing of reinforcements.* The closer the massing of the reinforcements, the greater will be the extent of the reminiscence effects (MOWRER and JONES, 1943, SOLOMON, 1948; OSGOOD, 1953).

(v) *The reaction threshold.* This will determine the speed with which the response is evoked.

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FRANKS (1956, 1957) has shown, for example, that the speed of conditioning tends to vary with the degree of introversion–extraversion.

(vi) *Further Hullian Concepts.* It is necessary to introduce two further Hullian concepts, namely those of reactive inhibition ($\overset{I}{(}R$) and conditioned inhibition ($S\overset{I}{R}$). According to Hullian theory learning, or the formation of stimulus-response connexions, results in varying degrees of fatigue or inhibition ($\overset{I}{(}R$), though the value of this inhibitory potential diminishes with rest. Stimuli which are closely associated with the acquisition of inhibitory potential ($\overset{I}{(}R$) become conditioned to it. This is known as conditioned inhibitory potential ($S\overset{I}{R}$) and tends to be much less affected by rest. Both the reactive and the conditioned inhibition summate functionally to produce the total inhibitory potential ($\overset{I}{(}R$). The reaction potential of the stammer (i.e. the ability of the subject to stammer) will therefore be determined by the following equation:

$$S\overset{E}{R} = (S\overset{H}{R} \times D) - (\overset{I}{R} + S\overset{I}{R})$$

As the $S\overset{I}{R}$ increases so should the $S\overset{E}{R}$ decrease, providing there is no increase in drive level (D). A point will eventually be reached when the excitatory and inhibitory potentials of the equation are the same and behavourial extinction of the stammering will have been achieved.

(vii) *A graded re-education programme.* This is designed for the purpose of substituting a normal reaction in place of the original anxiety reaction in the context of other people being present when he uses the telephone.

(b) *Deduction of method of treatment*

The above theoretical considerations are intended to show that certain cases of stammering can be conceived as specific types of learning or drive reducing conditioned avoidance responses. According to this model the stammer can be regarded as a simple learned habit which has reached maximum habit strength and which can be extinguished by building up a negative habit of not performing the stammer. This could be attempted in the following way. If the patient were asked to shadow the experimenter's voice and at the same time to speak into the telephone, then, according to the above theory just such a negative habit should develop. The habit of not stammering would then be associated with drive-reduction, because of the importance of this improvement to the subject, and so would be reinforced. Repeated massed practice would build up a negative habit and oppose the positive habit of stammering. The simultaneous use of the telephone and 'shadowing' would demonstrate to the subject that the telephone does not necessarily result in stammering. There will then be a corresponding decrease in the anxiety. As a result of the stress situation not resulting in the conditioned avoidance response, and because the stammering is itself influenced by the anxiety there will be a still further reduction in this avoident response.

A further decrease in excitatory potential should be achieved by the complementary effects produced by the graded retraining programme. The presence of people well known to him would constitute a weak excitatory stimulus, in contrast to the intense anxiety producing stimulus constituted by people at his place of employment. Since the weak stimuli are less likely to evoke an anxiety reaction the motivation towards achieving an alternative normal reaction is maximized. As the experiment progresses people less well known to him are introduced. Thus by increasing the differential stimuli intensity until a normal reaction supersedes the original anxiety reaction, involved in the original conditioning, a normal response is gradually achieved.

It is difficult to isolate the respective effects of the use of the telephone, 'shadowing', the conditioned inhibitory potential and the graded re-education programme. It appears that the excitatory potential of the equation will decrease and the inhibitory potential will tend to increase because of the gradual development of a negative habit and because of the decrease in anxiety associated with the re-education programme. The respective decrease and increase in excitatory and inhibitory potentials should eventually result in the extinction of the stammering. It is important to appreciate that as the decrease in excitatory potential becomes more marked the rate of growth of the inhibitory

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potential should become less. This is because less effort and difficulty will be experienced as the development of the habit of not stammering proceeds. The result should be a more regular and even level of performance as the experiment progresses.

(c) *Treatment procedure*

The internal telephone system of a neuropsychiatric unit was used. This made possible the simultaneous use of two internal telephones, so approximating the working environment of the patient.

Before the experiment began the technique of shadowing was demonstrated to the patient. One psychologist read from a book, the other shadowed, following one or two words behind. Following this demonstration three practice sessions were carried out in which one psychologist read from a book, the patient 'shadowed' him, and the other psychologist recorded any errors incurred by the patient during shadowing. An error was regarded as the inclusion of any word not in the original text or the omission of words from this text. During both the three practice trials and the subsequent trials the patient did not face the examiner.

After three trials the telephone was introduced. From trial 4 to trial 10 one psychologist rang the patient's telephone number, the patient answered and the psychologist proceeded to read the text to the patient. The second psychologist was in the same room as the patient and recorded any errors in the patient's shadowing. The patient listened to the first psychologist on the telephone then repeated by 'shadowing' what he had heard to the other psychologist.

Following the ten introductory sessions there was a reversal of procedure in that the psychologist read the text as before but this time to the patient in the same room. The patient 'shadowed' this material, repeating it directly into the telephone to the second psychologist in another room who immediately recorded the errors and hence the progress made.

In subsequent trials attempts were made to alternate the psychologist who was reading the text or recording the patient's progress. From trial no. 15 two other people were introduced into the experiment, one a member of the technical staff, the other a patient, and from then on one or other was invariably present during trials and whichever of these two people were present listened to and recorded the patient's progress. Both were well known to the subject. In later sessions two other people took part in the experiment. Neither of these people was known to the patient though they were introduced to him as relatives of one of the experimenters. Thus a graded re-education programme was devised based on a theoretical formulation proposed by GUTHRIE (1935) and successfully executed in clinical trials by WOLPE (1952, 1954). The original anxiety reaction to the stimulus of other people being present is replaced by normal reaction. By gradually decreasing the familiarity of the people in the experiment, though making sure that the initial stimuli are of a greatly reduced intensity the accustomed anxiety response is less likely to be evoked. As suggested by JONES (1956), '. . . the presentation of a stimulus qualitatively remote from the original, but occupying a position on the generalization curve of the latter, would operate in the same way as a reduction in stimulus intensity'.

RESULTS

Figure 1 shows the changes in frequency of stammering from the beginning to the end of the experiment. It will be seen that the introduction of the telephone in

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trial 4 resulted in an immediate increase in stammering, hesitations and inaccurate perceptions. This declined very rapidly only to rise again when the patient was asked to shadow directly into the telephone. This increase was very short lived. It was followed by a consistent improvement in telephone performance which is reflected by the downward trend in the graph.

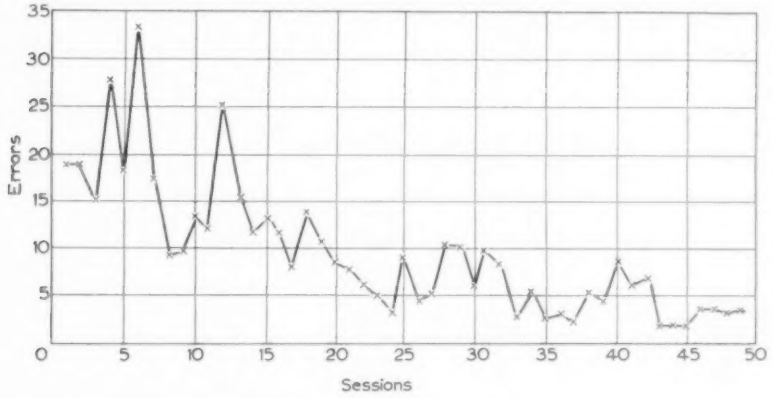

FIG. 1. Telephone performance measured by average number of errors (stammers, hesitations, misinterpretations) per page of text shadowed.

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Each figure quoted in the graph represents the mean number of stammers, hesitations and other errors per page of presented text. Sessions numbers 11–39 were 20 min in length, whilst trials 40–49 were 45 min massed-practice sessions. In a general way there appears no significant difference between the 20 and 45 min sessions, though it is not possible to establish this with any certainty because of the limited number of trials which were possible.* The results, however, are in accord with predictions and offer support for both the theoretical model proposed to explain the genesis of his stammering and the deductions for a rational method of treatment made possible from this theory. As a further check on the validity of the theoretical proposals and the methods of treatment following on directly from these, two additional criteria were required. First, it was necessary to see how well the patient performed on the telephone without the assistance of 'shadowing' and under conditions approximating to his working environment. In other words to see how well he had developed the negative habit or habit of not responding to the telephone by stammering; and the second criterion of success was whether or not the patient reported that he felt no apprehension with regard to the use of the telephone.

During the patient's final week in hospital there were twenty additional 10 min sessions. These consisted of one of the two psychologists ringing the patient's telephone and discussing with him subject-matter directly relevant to his job. The patient did not have the help of shadowing. His conversation was scored for stammers and hesitations. Conversations included discourses on uranium, and other elements important to atomic energy; isotopes; power; transformers; electrical relays; drawings; schedules; materials; and radiation effects.

* The experiment was started knowing the patient was due to emigrate to Canada, though sufficient time was available to test the general validity of the theory.

During these 10 min conversations it was difficult to obtain a strictly objective score as the patient talked for varying amounts in each session. As far as possible he was required to talk for the majority of the 10 min. The psychologist merely asked short relevant questions designed to keep the subject talking.

Figure 2 shows the number of stammers and hesitations per 10 min session over twenty trials. The graph reflects a striking decline in frequency as the experiment progressed. During the final trials of this series the stammers and hesitations were very infrequent. In fact the final ten trials with no shadowing showed an improvement over even the final ten trials with shadowing.

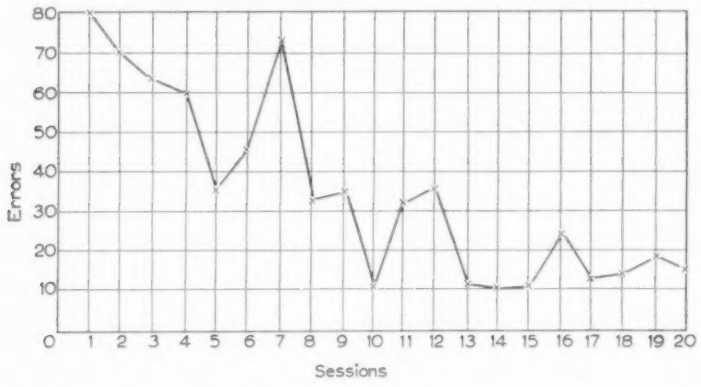

FIG. 2. Telephone conversation performance, without shadowing, measured by total number of errors (stammers and hesitations) per 10 minute session.

The day the patient was leaving hospital he was asked to write a frank account of his response to treatment. He reported, 'When I came into hospital I was extremely sceptical of success, having tried so many methods of treatment. In contrast to this, on discharge I am quite convinced that I have got into a healthier and better method of speech and am certain that my general speaking level has been maintained at its peak. Under treatment I have not had a bad patch (11 weeks). Prior to hospital I had a regular cycle of stammering during which 1 week in 4 was very bad, though at no time had I confidence in the telephone and I had fear of it irrespective of the week. Since the experiment I have had no such cycle and my level of general conversation has been consistently equivalent to my best week in the cycle. As regards the telephone I am still apprehensive towards it but I no longer consider it an insurmountable difficulty. I am convinced I can get over it and that my telephone conversation can be brought up to my conversational level. The shadowing may have been responsible for maintaining this high level but I have responded most I feel to the use of, and length of time using, the telephone'.

DISCUSSION

During the course of the treatments described, considerable improvement in the patient's major dysfunction was evidenced. The results would tend to support both the validity of the theory put forward to explain the genesis and specificity of his stammer, and the deductions for a rational method of treatment made possible from this theory.

It is not possible to state with absolute confidence that such improvement resulted from the treatment employed. Further controlled studies using similar methods of treatment would have to be carried out to determine this. One can say, nevertheless, that the present paper and those of several others (JONES, 1956; EYSENCK, 1956;

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YATES, 1957) demonstrate that it is possible to devise rational sympomatic methods of treatment for psychiatric symptoms, whether physical or emotional, that these symptomatic cures do not produce alternative symptoms (which should happen according to Freudian theory), that they are in fact long-lasting (WALTON and BLACK; JONES, 1956), and have been achieved when the patient has failed to respond to other psychotherapeutic methods over several years.

Several major problems have been presented by the present investigation. Although a satisfactory therapeutic result appears to have been achieved it was not possible in such an exploratory-experimental investigation to control and relate the different variables to specific therapeutic effects. Certain tentative conclusions can be made with regard to suggested optimum conditions of treatment.

First, the experiment would undoubtedly have benefited by a larger number of trials and reinforcements—'the increments from successive reinforcements summate in a manner that yields a combined habit strength ($S^{H}R$) which is a simple positive growth function of the number of reinforcements' (HULL, 1943).

Secondly it is possible that the experiment would have been further enhanced by longer lessons. The sudden introduction of massed practice following a series of shorter trials should then increase the inhibitory potential of the equation. At the same time the 'elation' effect, produced by success on the telephone over such a long period, would minimize the anxiety and so both decrease the excitatory potential and contribute to the development of the negative habit.

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It might also be more rapid therapeutically to concentrate first on only two treatment variables, viz: the decrease of the excitatory potential through shadowing and use of the telephone, and the increase of the inhibitory potential through massed practice. Thus one might leave the graded re-education programme until after the rate of experimental extinction had greatly decreased.

The patient for his part reported that he considered the continued use of the telephone therapeutically more helpful than the use of shadowing. One cannot decide, on the basis of the available evidence, whether this is so, though it would not appear to be the case, especially since inevitable and frequent use of the telephone in his present position has been his main difficulty. Experimental evidence outside the present work similarly suggests that repetition alone does not develop a habit either positive or negative but merely provides the opportunity for learning if a suitable reinforcement is present (HULL, 1943). The mere use of the telephone without shadowing would produce some extra degree of reactive and conditioned inhibition because of his difficulty and so reinforce the habit of not wanting to use the telephone. This would be expected to generate further anxiety which would in turn affect subsequent attempts to use the telephone. The simultaneous use of shadowing, however, prevents or minimizes the development of the anxiety since the previously learned response of stammering is less likely to occur. The use of the telephone alone, therefore, is likely to result only in an increase in inhibition and anxiety with little or no difference to the resultant of the excitatory minus the inhibitory potentials of the equation.

Finally it may be possible to integrate more closely learning theory, personality theory, incentives and stimulant-depressant drugs in their application to similar therapeutic problems. It will be remembered that FRANKS (1956) has shown that

introverted neurotics tend to condition far more easily than extroverted neurotics and that the latter group condition only with difficulty. In line with this formulation are the recent attempts by EYSENCK *et al.* (1957(a)–(e)) to relate the application of a stimulant drug (dexedrine) to more rapid learning by decreasing cortical inhibition (PAVLOV) or reactive inhibition (HULL) and so producing an introverted behavior pattern. It would appear reasonable to suppose that an initial series of shorter trials during which no drugs are applied, followed by a sudden increase in massed practice trials with dexedrine would approximate to the effects produced in CRESPI's experiment (1942). It may of course be better to sacrifice speed of learning by not using drugs, particularly if the patient feels that his symptoms have been removed artificially.

SUMMARY

The present paper attempts to formulate a theoretical model according to which the patient's stammer is regarded as a conditioned avoidance response; this response was originally evoked in an unknown traumatic situation and was drive-reducing.

A method of treatment was derived from this model. It was predicted that three major experimental conditions would build up the negative habit of not stammering when using the telephone. These were to decrease the excitatory potential, through the simultaneous use of the telephone and 'shadowing'; to increase the inhibitory potential through massed practice trials; and finally to substitute by means of a graded re-education programme a normal reaction for the original anxiety reaction to the telephone.

The results of the experiment tend to support the validity of the proposed model. The patient achieved a satisfactory level of performance on the telephone when unassisted by 'shadowing'. Furthermore, in a frank assessment of the effects of treatment, he himself reported considerable clinical improvement.

Various suggestions are made for the optimum growth of the negative habit. The proposals attempt to integrate recent experimental findings in learning theory, personality theory, incentives and in the use of stimulant-depressant drugs.

Acknowledgements—We are grateful to Dr. B. FINKLEMAN, Medical Superintendent, Rainhill Hospital for permission to publish the present study. Our thanks are due also to Dr. C. HELLER whose assistance made this study possible.

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BOOK REVIEWS

Theories of Personality. C. S. HALL and G. LINDZEY: John Wiley & Sons, New York, 1957. xi + 572 pp. 52s.

THE enormous spate of psychological literature continues to be a major problem for the clinician or research worker in a medical field. What to read and, still more important, what to believe—these are perplexing questions for the psychiatrist, let alone the physician or gynaecologist. To some extent the psychologists are themselves to blame for this state of affairs: they write too much, the number of schools is reminiscent of eighteenth century medicine, and indeed some of the polemic might be taken straight out of that hard-hitting period. The fact that psychiatry is often belaboured makes for bad blood between the two disciplines most likely to be of help to the physician in the field of psychosomatic medicine, and certainly one result is that psychology has not yet come to take its rightful place in the team. The evil effects of this state of affairs are nowhere better seen than in the present confused state of personality study in psychosomatic disorders. Just how naive much of this work is can at once be seen after reading this excellent book, the purpose of which is to present a summary of contemporary theories of personality.

Beginning with a brief survey of the nature of personality theory, the authors describe the theories of FREUD, JUNG, MURRAY, LEWIN, ALLPORT, of Social Psychological Theories, Factor, and Stimulus—response theories, CARL ROGERS and other contemporary workers. An interesting and useful, although brief biography of each major theorist is given, and this is followed by a well organized and comprehensive account of the particular theory. Professors HALL and LINDZEY write admirably, and express their own views moderately and with a charming courtesy. The book is essential to all those concerned with the study of personality.

DENIS LEIGH

Ciba Foundation Colloquia on Endocrinology. Vol. 10. **Regulation and Mode of Action of Thyroid Hormones.** Edited by G. E. W. WOLSTENHOLME and E. C. P. MILLAR. J. & A. Churchill Ltd., London, 1957 xii + 311 pp. 48s.

IT is never easy to review the proceedings of a colloquium. To comment upon one paper means that another must be neglected, whilst a brief note on each contribution produces the equivalent of an annotated bibliography. This volume provides no exception to the rule. Here we have eighteen authoritative papers covering aspects of thyroid function as diverse as its role in immune responses, the metabolism of thyroid hormones by the kidney and the regulation of thyrotrophic hormone secretion by the nervous system. It may be noted that the range of contributions extends beyond that indicated by the title. Few readers will expect to grasp all the information presented at this meeting, particularly as the numerous abbreviations for the products of the thyroid gland appear rather formidable and are prominent in its pages. However, those concerned with psychosomatic problems will find the papers by BROWN-GRANT, COURRIER, GREER, and HARRIS and WOODS on the neural control of thyroid activity among those more easily assimilated. In general, this volume provides a useful summary of some features of thyroid gland physiology which are undergoing rapid development and fully maintains the high standard of its companions in the series of Ciba Colloquia on Endocrinology.

B. T. DONOVAN

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The Neurohypophysis. Edited by H. HELLER. Butterworths Scientific Publications, London, 1957. xvi + 275 pp., 50s.

UNTIL quite recently the study of neurohypophysial function has tended to be unfashionable. Experimental work on the gland is somewhat difficult because of its position at the base of the brain and, in any case, its products do not exert such spectacular effects as those of the anterior lobe of the pituitary. For such reasons, progress in this field was slow until interest was aroused in the secretory activities of certain nuclei associated with the organ—in neurosecretion—and it became apparent that antidiuretic hormone and oxytocin might be concerned in the control of anterior pituitary activity. Now, with so much attention being given to the gland, progress is likely to be rapid. The present symposium was therefore held at a most opportune time and provides a valuable survey of current thought in this field.

The volume opens with an historical introduction by Sir HENRY DALE and continues with papers on neurohypophysial structure, neurosecretion, and the nature and metabolism of neurohypophysial principles. The actions of the hormones on the kidney, uterus and mammary gland next receive treatment, and the symposium closes with an examination of the adrenal–neurohypophysial relationship. As is customary at the symposia of the Colston Research Society, each paper is followed by a discussion, which often proves most illuminating.

Whilst the classical actions of the posterior pituitary hormones are emphasized, newer concepts, such as the connection between oxytocin release at mating with sperm transport and the presence of a drinking area in the anterior hypothalamus have not been neglected. Neurosecretion and the possible integration of anterior and posterior pituitary function are dealt with at some length. It will be seen that this symposium provides a broadly based and extremely useful survey of recent work on the neurohypophysis, and the organisers, and in particular Professor HELLER, are to be congratulated on the success of their efforts.

B. T. DONOVAN

Neurochemistry. Edited by S. R. KOREY and J. I. NURNBERGER. Cassell, London, 1957. 244 pp., 50s.

THIS is the first of yet another series of symposia, to be published under the general title of *Progress in Neurobiology*. The present volume contains some 16 essays by as many authors on diverse topics in brain biochemistry, physiology and, in one instance, anatomy. Despite a uniformly high standard in the individual contributions the reader seeking a comprehensive background to a rapidly developing subject will be disappointed; on the whole this is very much a specialist's book. Most of the papers are accounts of the authors' own work, often about methodological problems, and generally written in a style more suitable for journal publication. It is, moreover, unfortunate that in compiling a book entitled *Neurochemistry* the Editors have found it necessary to include several papers, excellent in themselves, but concerning subjects with little relevance, at least in our present state of knowledge, to brain biochemistry. The result is a book lacking cohesion and unity of purpose to a degree exceptional even for a symposium, admittedly a species where these faults are rarely altogether avoided.

Fortunately the defects possessed by the book as a whole are compensated by the inclusion of an excellent review entitled "The Neurochemistry of Seizures" by D. B. TOWER, which deals very clearly and at considerable length with the evidence for an abnormality in acetylcholine and glutamate metabolism in epilepsy, and a very interesting paper by NURNBERGER and GORDON on "Effects of Brief Stress on the Ribonucleic Acids and the Labile Nitrogen Pool of Brain and Liver in the Rat." For clinically orientated readers there are also papers on ceruloplasm and plasma copper levels in Wilson's disease (SCHEINBERG) and on brain lipoproteins in allergic encephalitis (GOLDSTEIN and KIES).

R. RODNIGHT

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RAYMOND J. CORSINI: **Methods of Group Psychotherapy.** McGraw-Hill, New York, Toronto, London, 1957. 251 pp. $6.50.

THIS is the most comprehensive and enlightening book on the subject of group psychotherapy that has appeared so far. Interest in group psychotherapy has soared since 1940 and there have been more than 1700 publications since then. Most of the authors, however, have been content to plough a lonely furrow of their own design without casting more than an occasional critical and deprecating glance at the parallel efforts of some other plough-men in the field. It seems that group therapists, by and large, are immune to those cohesive forces which are operative in most other groups of people with a common aim. In spite of these tendencies there have nevertheless been a few schools of thought whose leaders have evolved their own terminologies and have been untiring in stressing the superiority of their own methods.

The author of this book has tried to unravel some of the resulting confusion of language and thought by equating apparently synonymous terms in the vocabulary of different group therapists (e.g. reassurance = ego support; re-education = insight), and by reducing the 166 separate group dynamisms described in the literature to a semblance of order by categorizing the majority of them as being emotional, intellectual or behavioural in quality. Into the emotional category he has put such processes as tolerance and sympathy which appeared in the literature under a multiplicity of deceptive names; into the intellectual category such phenomena as the realization and understanding of shared symptoms; and into the behavioural category such ideas as the testing of a social reality through interaction and communication with others.

He has used a similar simplifying scheme in an attempt to classify the many methods employed by different group therapists. He has divided the therapists themselves into a directive and a non-directive camp. The directive therapists, according to the author, tend to be forceful, rational and didactic; the non-directive therapists, on the other hand, are inclined to be patient and passive.

Many directive therapists use Adlerian methods of counselling to show their patients what is wrong with their reactions and how they can alter their lives. Others use such methods as the authoritarian "will training" of A. A. LOW, diverse lecture methods, the repressive-inspirational techniques of MARSH and others, the psychodrama of MORENO, group bibliotherapy, and even a "mechanical group therapy" which needs neither therapist nor group but only short recorded messages played over the loudspeaker system of a mental hospital.

The non-directive therapists use subtler means to achieve their ends. The psychoanalysts among them interpret the noxious motivations behind their patients' symptoms. The client-centred followers of CARL ROGERS do not even go as far as that; their patients explore their own motivations in an all-permissive atmosphere of non-critical acceptance. Among other methods mentioned under this heading are leaderless groups, social clubs, activity groups, music therapy, and many others.

The author also presents some invented typical examples of group sessions which illustrate the working of family counselling, psychodrama, analytic interpretations, and client-centred non-directiveness.

Not only neurotic, psychotic and psychopathic persons have been treated with group methods but also patients suffering from such organic conditions as tuberculosis, diabetes, hypertension, epilepsy, disseminated sclerosis, aphasia and senility. Group therapy is supposed to help them to adapt to their physical disabilities. Psychosomatic diseases, such as peptic ulcers, allergies and obesity, have also been group-treated.

After the evidence of so much ingenuity in inventing group theories and practical methods that can be applied to an ever-widening circle of diseases, it comes as a disappointing anti-climax to see that the chapter on research contains only few references. The research that has been done on therapeutic evaluation is summed up by the author like this: "The statistics in reality prove nothing but . . . . that the therapists who report them appear to find them impressive".

F. KRAUPL TAYLOR

R. S. WALLERSTEIN *et al.*: **Hospital Treatment of Alcoholism** Imago Publishing Co. Ltd., x + 212 pp., 42s.

THE treatment of alcoholism is still the despair of psychiatrists, although much therapeutic effort has been expended on these patients, particularly since the end of World War II. The fact that A.A.

seems to provide by far the best hope for the alcoholic alone is evidence of this therapeutic failure. Nonetheless, proponents of one or other method of treatment—deconditioning, antabuse, hypnotherapy and latterly, group therapy—are not hesitant in putting forward their claims. In order to assess the usefulness of these major treatments a project was set-up by Dr. WALLERSTEIN and his associates, in which the three methods (antabuse, conditioned reflex therapy, group hypnotherapy) could be subjected to detailed analysis, and more important still, a control group was used. The number of hospitalized alcoholics, studied over 2½ years of treatment and 2 years of follow-up, was 178.

The results showed that antabuse therapy was the most useful, but even so half of the group receiving antabuse did not benefit. Similarly some patients in each of the other groups responded to the particular treatment, others not. A study of the reasons for success or failure seemed to be most related to the interaction of the personality of the patient and the type of therapy—for instance compulsive characters did better with antabuse, the threatening punishing nature of the conditioned reflex therapy seemed to relate to the patients aggressivity or need for punishment. Unfortunately as with so much work on treatment in psychiatry, the results are unimpressive, and the project in fact, serves only a suggestive purpose. Some might wonder why an investigation of this type need appear in book form, another addition to the vast numbers of books masking our ignorance. But it is a useful book for both the general physician and the psychiatrist who has no special interest in alcoholism, and is a useful addition, at least on their book-shelves.

DENIS LEIGH

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Papers to be published in future issues

E. GELLHORN: Recent contributions to the physiology of the emotions.
M. GERBNER and K. ALTMAN: On the mechanism of the diuretic conditioned reflex.
M. GERBNER, K. ALTMAN and I. MISZIROS: The mechanism of the increase in diuresis induced by hypnotic suggestion.
T. G. KERN, J. H. EWING and K. RICKELS: The hippuric acid index: application of the hippuric acid test as an index of humna free anxiety.
S. I. COHEN and A. J. SILVERMAN: Psychophysiological investigations of vascular response variability.
LINFORD REES: An evaluation of the role of emotional factors in miners' nystagmus.
LINFORD REES: The role of emotional and allergic factors in hay fever.
D. WALTON and D. A. BLACK: The application of modern learning theory to the treatment of chronic hysterical aphonia.
CAROL BUCK and G. E. HOBBS: The problem of specificity in psychosomatic illness.
M. GERBNER, K. B. KOVACS and K. ALTMAN: On the glomerular factor in water diuresis.
M. GERBNER, K. B. KOVACS and K. ALTMAN: The effect of adjustment to the environment and its alteration with water diuresis.
CHARLES SHAGASS, KARLA MULLER and B. ACOSTA: The pentothal sleep threshold as an indicator of effective change.
BRIAN ACKNER and G. PAMPIGLIONE: An evaluation of the sedation threshold rates.

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ERRATUM

Delete the paper E. GELLHORN: Recent contributions to the physiology of the emotions.

J. Psychosomatic Res., 1959, Vol. 3, pp. 185 to 210. Pergamon Press Ltd. Printed in Northern Ireland

PSYCHOPHYSIOLOGICAL INVESTIGATIONS OF VASCULAR RESPONSE VARIABILITY*

S. I. COHEN, M.D., and A. J. SILVERMAN, M.D.

(*Received* 3 *March* 1958)

INTRODUCTION

THIS paper will describe an approach to the study of psychogenic influences on vascular phenomena. The approach was concerned primarily with the investigation of the alteration of response to physical and physiologic stimuli which affected the cardiovascular system. Previous studies have indicated that emotional factors may influence the vascular system, and it appears likely that this influence is mediated primarily through the autonomic nervous system and/or neuro-hormonal factors.

The response to a stimulus affecting the vascular system should involve the activation of reflex mechanisms to compensate for the changes initiated by the stimulus. Emotional factors might then influence vascular response patterns by affecting the reflex circuits through neural or hormonal pathways.

The area chosen for study was the variations in response to a stimulus producing 'hypotension' in the head and neck area. It appeared logical to assume that this would initiate reflex changes set off in the carotid sinus, aortic arch, etc., which would be aimed at increasing the effective arterial pressure in the head and neck. The effect of emotional factors could then be assessed by evaluating alterations in the compensatory response to stimuli producing these hypotensive effects.

Most of the work which was done and which will be described subsequently in this paper involves the use of two types of stimuli affecting the cardiovascular system. Firstly, subjects were exposed to the effects of increased gravitational forces by making them ride a human centrifuge capable of reaching high levels of acceleration in which increased gravitational forces were exerted in a head to foot direction inducing effectively a drop in pressure in the head and neck as a result of the pooling of blood in the periphery. Secondly, subjects were given small doses of mecholyl subcutaneously. This parasympathomimetic drug is believed to have a primary vasodilatory action on peripheral blood vessels. Since the subjects would be kept in a seated position when the drug was administered, it was anticipated that the vasodilatation produced by the drug plus the effect of posture and immobility would lead to a decrease in pressure in the head and neck region.

The experimental hypotheses were concerned with delineating the effects of specific emotional states on the reflex circuits activated by the above mentioned stimuli and an enumeration if possible of the mechanisms by which these effects were mediated.

The work of others (ACKERMAN, 1950; ACKNER, 1956a,b; ALEXANDER, 1939; BINGER *et al.*, 1951; ENGAL and ROMANO, 1947; FERRIS *et al.*, 1948; PFEIFER *et al.*, 1950; REISER and FERRIS, 1947; SASLOW *et al.*, 1950; SAUL, 1939; STEAD, 1945; WOLF *et al.*, 1948) suggested that the arousal of emotional factors in experimental

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and life situations was associated with changes in blood pressure, as well as being involved in the etiology of or the exacerbation of hypertension. In general, most of the findings suggested that angry or hostile feelings or aggressive impulses, thoughts or behaviour were involved in the vascular changes which were noted. It had also been noted that anxiety appeared to be associated with changes in cardiac functioning as well as vasomotor tonus. The work of other investigators (AX, 1953; FUNKENSTEIN *et al.*, 1952, 1953, 1954a,b; RIOCH, 1956; SCHACHTER, 1957) was more specifically focused on the delineation of the role of anxiety, anger and depression in various types of vascular responses. It appeared from the work of these investigators that anxiety was associated with an increase or decrease in systolic pressure but frequently with a decrease in diastolic pressure, and it was speculated that anxiety was related to the release of adrenaline, probably from the adrenal medulla, which led to a peripheral vasodilatation and a lowered peripheral resistance, but that the cardiac rate and stroke volume were increased. However, it was felt by these investigators that 'anger out' may be associated with the release of noradrenaline like substances with an increase in peripheral vasoconstriction and consequently an increase in peripheral resistance.

Possible central mechanisms for these responses were suggested by the work of others (BARD, 1940, 1950; CHAPMAN, 1950; FOLKOW and VON EULER, 1954; GELLHORN *et al.*, 1955, 1956a,b; HESS, 1949). The work of these investigators suggested several factors. First, it appeared that cortical, as well as hypothalamic areas, were capable of producing alterations in blood pressure when directly stimulated. In addition, it was also suggested that the anterior and posterior hypothalamus might exert a tonic effect upon the medullary vasomotor regulating centres. Stimulation of the hypothalamus by FOLKOW and VON EULER (1954) revealed that there appeared to be specific areas which when stimulated, led to the release of adrenaline in the peripheral blood and urine, while other areas led to the release of noradrenaline. Whether these areas were related to hypothalamic areas which were felt to be associated with anxiety and rage responses is still a moot question. However, the evidence did suggest that at least the adrenaline-producing areas of the hypothalamus were similar to those portions which had been previously identified as centres for fear responses.

Although it is still speculative, it is strongly suggested that there may be central mechanisms mediating specific emotional responses such as fear and anger, and that these mechanisms may be related to neuronal circuits concerned with the release of catechol amines, as well as the activation of portions of the autonomic nervous system.

I. RESPONSE TO THE HUMAN CENTRIFUGE

The human centrifuge at Wright Patterson Air Force Base is capable of accelerating subjects up to 22 *g*. In the experiments conducted in this study, the gravitational pull was exerted in a head to foot direction (positive *g*). When the *g*-force is sufficient to reduce the effective pressure in the head and eyes below the intraocular pressure, peripheral vision and then central vision is lost. The loss of central vision is known as blackout. Following this, if the pressure continues to drop, the subject may become unconscious. With the onset of positive *g*, there is a brief fall in arterial blood pressure. This fall is enough to act as a stimulus for various baroreceptor systems. Pressor receptors in the carotid sinus, aorta, and the heart itself

are activated and a chain of compensatory cardio-vascular reflexes is initiated. The result is seen in reflex changes, in heart action, the arterial tree and in the venous system. Blood pressures measured at heart level show a rise while the subject is under *g*. However, this rise is misleading because of hydrostatic pressure differences at heart and head level. Direct arterial pressure measured at heart level shows an abrupt rise as compensatory reflexes exert their effect. But there is always a net increase in arterial pressure at head level. Blackout occurs when the neck pressure is exceeded by the intraocular pressure. The vasomotor reactivity of the arteries and veins is important in determining tolerance to acceleration. (EDELBERG, in press; EDELBERG *et al.*, 1956; HENRY and PEARCE, 1956; SALZMAN and LEVERETT, 1956; ZUIDEMA *et al.*, 1956b). The degree to which vasomotor tone can be increased varies considerably from one subject to another, but it is of overall importance two ways: (1) Arterial responsivity determines to what extent the peripheral resistance can be increased and this in turn has a direct bearing on effective arterial pressure at head level. With lowered peripheral resistance, blackout occurs at a lower level for at this point blood pools faster and blood pressure recovery is low. (2) Venous responsivity is important in maintaining cardiac output. Two-thirds of the total circulation blood volume is on the venous side (SALZMAN and LEVERETT, 1956).

Further evidence of the relationship of vasomotor reactivity in determining *g*-tolerance is seen in the relationship of post valsalva overshoot of arterial blood-pressure and *g*-tolerance. BONDURANT (1957) studied this relationship which appears to be a simple but quite reliable measurement of autonomic tone. A highly reliable (cc 0·89) relationship was found to exist between *g*-tolerance and the val-salva overshoot just prior to exposure to centrifugation.

Work by GOODALL and MEEHAN (1956) suggested that sympathetic responsivity and adrenal medullary discharge may both be involved in influencing vasomotor tone during centrifugation. Their work suggested that *g*-tolerance was higher in subjects who excreted the highest levels of noradrenaline in their urine following the runs. The noradrenaline liberated was felt to represent the noradrenaline released at the sympathetic neurovascular ending. Furthermore, adrenaline levels seem to be of little importance in influencing *g*-tolerance (vasomotor tone) when the noradrenaline was high. However, in the presence of low noradrenaline levels an increase in adrenaline might be more effective and lead to peripheral vasodilatation and a decreased tolerance to *g* in a person who is seated in a relatively immobile position.

If emotional factors affected vascular responses through autonomic or neurohormonal mechanisms, then it seemed plausible to assume that variations in vascular tolerance to gravitational forces could be produced or modified by emotional factors. This idea was supported by anecdotal data (SILVERMAN *et al.*, 1957) which suggested that persons who were depressed, sad, anxious have a low tolerance to *g*-forces on the human centrifuge or while flying high speed aircraft. On the other hand, persons who felt aggressive, angry, or appeared to be in control of a situation or were able to master a situation comfortably seemed to have higher *g*-tolerances. In addition to the inter-individual variability which is suggested above, an intra-individual variability was noted. Previous studies attempting to more specifically clarify the reasons for the inter- and intra-individual variability and susceptibility to gravitational forces were not successful in correlating physical or physiologic parameters with the variations.

A. Focused projective tests (FTT), specific personality features and average g-tolerance.

Observations on volunteer human subjects revealed that in addition to the individual day-to-day variations in *g*-tolerance, certain persons were predominantly capable of tolerating high *g*-levels while others could be characterized as low *g*-tolerance people. That is, one group of subjects seldom blacked out below 3·7 *g* and others seldom reached 3·7 *g* without blacking out. Naturally, some subjects showed considerable variations and had blackouts in both ranges. Nevertheless, it was striking how certain persons consistently had high-*g* tolerance while others were rather consistently low-*g*. If a psycho-physiologic relationship existed, then it was hypothesized that consistent high-*g* subjects might show persistent personality patterns which differed from consistent low-*g* subjects. The personality characteristics which were felt to be related were, the affect state, that is level of anxiety, level of anger and the usual behavioural mode, particularly as related to the direction, type and intensity of aggressive expression.

Initially, it was felt that existing clinical psychologic tests were aimed at obtaining too global a psychologic picture to be of any great value. However, as will be indicated later, and has been described in other papers (SILVERMAN *et al.*, 1954) this was not a completely valid assumption.

The first step taken was to design a 'thematic apperception test' which was loaded with aggressive symbols and situations. The cards were presented and the stories given by the subjects were graded by criteria which were focused on such factors related to the handling of aggressive impulses. Although the test was designed to also grade the level of anxiety, it soon became apparent that the test had its primary usefulness in elucidating the projections of the subject's aggressive behavioural patterns and his comfort and control in this area.

Subjects with high average *g*-tolerance (that is consistently over 3·7 *g*) scored high in the ability to express, anger and impulsive actions, and they appeared comfortable in doing this, that is the aggression was expressed without guilt, anxiety, shame, or fear. Furthermore, the heroes in the stories were persistent and able to master the situations described.

Consistent low-*g* subjects told stories with little activity, about heroes who were passive, who identified with an aggressed against person and in which the hero was not persistent, impulsive or able to master the aggressive situations portrayed in the cards.

Four studies using the focused thematic test were done (SILVERMAN *et al.*, 1956, 1957a,c,d) the last two being double blind studies using subjects whose *g*-tolerance was not obtained until after the test was scored. Following the test, they were given a series of weekly rides in order to obtain an average *g*-tolerance. All in all, twenty-six of thirty-two low-*g* subjects were correctly placed and forty-six of fifty-two high-*g*. These figures were significant at better than a 0·03 level of confidence. It was later noted that the Rorschach and standard TAT stories could be used successfully to place the subjects if focused criteria were utilized (COHEN *et al.*, in press). The level of personality integration did not reflect the capacity to tolerate *g*-forces, except in so far as it determined what stresses a person will respond to and how he might react. The ability to express aggressive thoughts comfortably in the stories appeared to be the most important criteria differentiating the persons capable of resisting different intensities of vascular stress.

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B. Level of anxiety and average g-tolerance

Although many tests have been designed to give a picture of the level of chronic anxiety, no really satisfactory test appears to be available. In part this may be a function of the uncertainty about the definition of anxiety and the psychologic and physiologic processes which should be considered as a part of it. As will be pointed out later, the anxiety level immediately after an acute experiment was evaluated by a focused interview technique which was relatively successful in assessing correctly those persons with vascular responses to centrifugation which would be expected in persons with high anxiety levels. However, the problem of a test to assess the chronic anxiety level was not solved. Some work has been done using the level of

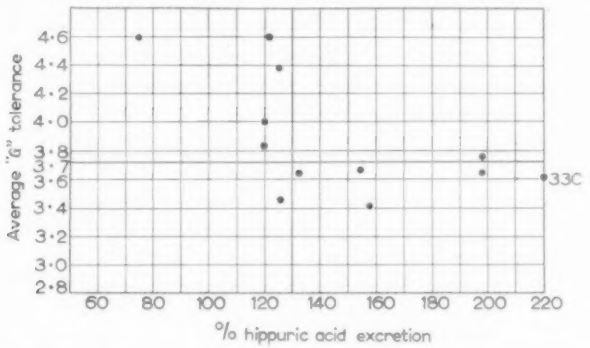

FIG. 1.—The hippuric acid excretory rate of the subjects is plotted as the percentage of normal excretion. The vertical line indicates the cut-off point of 130 per cent which was chosen to represent the upper limit of normal. The *g*-tolerance plotted is the average *g*-tolerance over a year's period in subjects whose tolerance did not vary more than 0·4 *g* during this period. The horizontal line represents the division of high- and low-*g* using 3·7 *G* as a cut-off point.

a hippuric acid excretion after the injection of sodium benzoate as a reflection of chronic anxiety (PERSKY *et al.*, 1952, 1954). PERSKY *et al.* have shown that a hippuric acid excretory rate in persons who chronically showed high levels of anxiety was higher than those who did not. Furthermore, the rate did not appear to be altered by acute or focal stress or acute anxiety, it was a function of chronic anxiety. Repeat levels in any of these subjects were rather consistent. PERSKY (1952, 1954) believes that ACTH and adrenocortical hormones, released in response to distressing stimuli may lead to a depletion of tissue glutathione (gluta-thione is the glycine donor which is involved in the detoxification of sodium benzoate and may lead to increase in hippuric acid excretion).

Twelve of the volunteer centrifuge panel were given 6 g of sodium benzoate dissolved in 30 cm^3 of water flavoured with peppermint. The normal 4 hr excretion is 4–5·3 g. (PERSKY, BASOWITZ, GRINKER, *et al.* utilized an I.V.-administration of 1·77 g and a urine determination in 1 hr in their studies. In the present study, the urines were collected hourly for 4 hr.) The percentage of normal excretion was then determined. A level lower than 70–75 per cent indicated liver disease. However, a level over 130 per cent normal was believed to be a function of high chronic anxiety. The twelve subjects chosen were consistently above 3·7 *g* or below 3·7 *g*. Five of the six high-*g* subjects had excretion rates less than 130 per cent. One low-*g* subject had excretion rates of 128 per cent. Five of six low-*g* subjects had excretion rates

above 130 per cent. One high-g subject had an excretion rate over 130 per cent (his average g-tolerance was 3·72). These preliminary results (Fig. 1) suggest that the hippuric acid excretion rate reflects in a general way the average vascular response characteristics of subjects to the human centrifuge. Furthermore, studies by other authors had already suggested the relationship of the anxiety level and the hippuric acid excretion rate. Hence, it was concluded that the level of free floating chronic anxiety may in some way reflect the individual's ability to tolerate g-forces over a period of time.

C. Affect state, g-tolerance and catechol amine levels

In order to more directly assess the relationship of emotional factors, the response to centrifugation and the possible role of catechol amines, a group of six subjects were given a series of centrifuge rides to establish the g-levels they were able to tolerate before blacking-out. Urines were collected before the centrifugation and immediately after centrifugation and at 1 and 3 hr after the post-run specimen. These urines were sent to another laboratory where they were bioassayed* for adrenaline and noradrenaline levels. Immediately following the run, the subjects were interviewed by a psychiatrist. The primary focus of the interview was to establish the affective reaction of the subject to the stresses inherent in the centrifuge situation. This included evaluating the anxiety level on a 0–3 plus scale and the aggressive thoughts, feelings and impulses engendered during the experiment on a 0–3 plus scale. The technique of the interview and more details about results of these interviews have been described in previous papers (COHEN *et al.*, 1956). The three parameters (affect state, catechol amine levels and vascular responses, i.e. blackout level) were assessed by independent observers without knowledge of the findings of the other experimental team. The details of this experiment are discussed in other publications (COHEN *et al.*, 1956, in press; ZUIDEMA *et al.*, 1957).

It should be pointed out that aggression was not used as a substitute term for anger or hostility. It was specifically defined as the active pursuit of desired objects or events or the active removal of frustrating or unpleasant situations. That is, active attempts made by the subjects to master or control situations imposed upon them. This might or might not be associated with the feeling tone of anger. Hence, it is obvious that what was actually being evaluated was (1) a feeling tone of anxiety while the other parameter was a combination of a behavioural mode (aggression) and possibly a feeling tone, anger or hostility.

The three subjects who were judged to be markedly anxious (3 plus) and whose aggressivity rating ranged from 0 to 2 plus all blacked-out at below 3·6 g. Two of these subjects were in a state of acute anxiety when they were interviewed following the centrifugations, and these subjects blacked-out at 3·1 and 3·3 g respectively. These were the lowest blackout levels recorded in the series. The three subjects with an aggressivity rating of 3 plus and an anxiety level of from 0 to 2 plus blacked-out at levels over 3·9 g and usually over 4 g. The blackout levels and the psychiatric evaluations of all six subjects were consistently related.

The urinary adrenaline levels were the highest in the low-g subjects in both the pre-experimental control specimens and the specimen collected immediately after

* The urinary bioassays were carried out by Dr. MCCHESNEY GOODALL at Duke University School of Medicine.

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centrifugation. The noradrenaline levels were highest in subjects who were able to tolerate higher *g*-forces. The noradrenaline findings were the most striking in that there was almost a straight line increase in noradrenaline levels following the centrifugation as one moved from the low to the high *g*-tolerance subjects; Fig. 2 indicates the adrenaline and noradrenaline levels before and after centrifugation in subjects blacking out at various *g*'s. It is interesting that the highest *g*-subjects in general

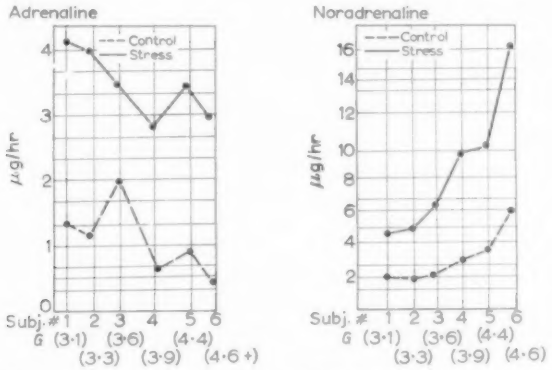

FIG. 2.—The pre-run adrenaline and noradrenaline levels of the subjects is indicated by the broken line and is plotted in terms of micrograms per hour. The subjects are numbered and their *g*-tolerance is noted. The solid line represents the post-run urinary catechol amine level.

have the lowest control levels of adrenaline suggesting that they were much less anxious prior to the experiment. In addition, their adrenaline output following centrifugation was lower. The converse was true of the noradrenaline levels. It was higher in the control specimen and certainly higher in the run specimen.

D. Altered affect state and g*-tolerance*

In order to further demonstrate or more definitely establish the relationship of *g*-tolerance and affect state, it was felt necessary that the experiment be repeated with an attempt made to alter the affect state of the subject and then determine if the blackout level and catechol amine level also changed. Five subjects had repeat experiments in order to determine whether *g*-tolerance shifts were associated with affect changes. The second experiment was kept as similar as possible to the initial experience. In two of the subjects, direct attempts were made to alter the affect state. But in the other three, the aim was to re-evaluate in the same manner the response to the experimental situation, without any active attempts to alter the affect state. In both cases attempts would be made to determine if the triple corelation noted in the first experiment was consistent.

In the two subjects where direct attempts were made to alter the affect state, the experimenters tried to push the emotional reaction in a specific direction. However, it was realized that no *a priori* assumption could be made regarding the subject's response. Hence, the final evaluation of the affect state during the second experiment would again be left to the focused interview to be held at the end of the centrifuge rides. An example of the methods used to alter the subject's overall emotional state is illustrated below.

The subject had blacked-out at 3·1 *g* in the first series (low-*g*). In general he had a record of usually being low-*g*, although on occasions his blackout level was over 3·7. Previous controls had established that he was an insecure chronically anxious and often extremely self-punitive person. Since his *g*-tolerance was low it was decided to raise it or attempt to raise it by provoking him into an aggressive or perhaps angry but not anxious state. However, in the past it had been noted that anger for this subject was extremely anxiety provoking. This was in part due to self-imposed, rigidly high standards of behaviour. Raising one's voice or even feeling anger, to him was an indication that he was failing to obey internal prohibitions. Instead of getting angry he would feel sorry for those who were treating him unjustly. It was considered that perhaps anxiety-free anger could be aroused if a respected authority gave him permission to be annoyed. It was further felt that under these circumstances the subject, being justifiably aroused to anger, could handle himself in the centrifuge situation with more assertive aggression and this might allow him to safely displace the anger which he usually found difficult to express directly. It so happened that the subject was in charge of scheduling the time for centrifuge experimentation, a job which at times was trying for him because of the impossibility of satisfying all the demands. This situation suggested the psychologic stimulus to be used. The subject's respected chief was instructed to meet the subject 'accidentally' just prior to the experiment and to provoke him as follows: The subject was greeted by the chief warmly and in general supported by the chief who however appeared angry and tight-lipped. The chief admitted to the subject that he was extremely angry with the experimental team because of their continual grumbling that the subject wasn't scheduling enough time for them on the centrifuge. He pointed out to the subject that he had every right to be angry and that he as the chief was furious because he knew the accusations were false. He knew that the subject was doing a good job, and without involving the subject in any way, was going to see to it that the experimenters learned the error of their ways. Apparently the plan worked. First the subject became a little upset and a little sorry for those who were critical of him but as the minutes sped by and as the chief continued his exhortations he gradually appeared less anxious and finally expressed his verbal annoyance which was continually supported by his supervisor. After 10 min although still somewhat anxious he appeared considerably angry. In the interview following the second series of experimental rides, the subject stated that although he had some tachycardia it didn't concern him as much as in the previous rides. He said that he felt cooperative during the experiment but was somewhat irritated when the people during the experiment told him to sit still. After all he had been trying to sit still during the whole run. (During this series of experiments as well as the previous series all the subjects were chastised from moving about at a time when the records indicated they were sitting still. The purpose of this chastisement was not only to provoke some affective reaction but the stimulus was to be used as focus around which the interview following the centrifugations would be concentrated in order to evaluate the subject's affective response). He had verbally responded to the chastisement presented at the beginning of the experiment stating that he had been sitting still. He directly verbalized the fact that he felt less anxious during the experiment and more comfortable and this time the *g* actually felt good. He then spoke quite freely about the sense of competition that existed between his research unit and the research unit of the investigators. He indicated that he felt pretty good about the competition. He then said that he was somewhat critical of the experimenter's research in attempting to categorize people by their blackout because he really didn't think that he was low-*g* and he was going to prove to us that he wasn't. During this interview, the subject appeared much more integrated and less anxious than during the previous interviews. He was much more comfortable in discussing his feelings of irritations at being chastised and he was more sure of himself. Although he had momentarily been irritated in the first experiment he had become apologetic about it and the aroused anger was apparently turned against himself. Self incrimination was strongly evident in the first experiment, at least in the post-run interview and totally absent in this one. Thus, it seemed that the somatic evidence of anxiety was much less striking during the second interview as well as the verbal content. The subject was outgoing poised and seemed to be better integrated. He was judged to be 3 plus aggressive and 2 plus anxious as compared to the first series where he was 3 plus anxious and scored zero aggression. Because of the decrease of anxiety and the increase in aggression his *g*-tolerance was estimated as middle or above middle, i.e. 3·7 or slightly above. The actual *g*-tolerance of the subject (noted on the following chart) in this series of runs was 3·8 *g* which is considerably higher than the first series which was 3·1 *g*. The stress catechol amines for this subject during the first series was 4·2 μg/hr of adrenaline and 5·5 μg/hr of noradrenaline. Following the

second series the stress adrenaline was again about 4·2 but the stress noradrenaline had risen to about 9·4, an increase of about 4 μg.

In a second subject who was also angered by the specific affect stimuli, his *g*-tolerance rose from 3·6 to 4 *g*. Of the other three subjects in whom attempts were not made to alter the affect state but rather to re-evaluate the correlation which existed when they were re-exposed to the same experimental situation, two of the subjects revealed altered *g*-tolerance. One subject had a lowered blackout level and his post-run interview revealed an increase in anxiety and a decrease in aggression

Subject	Experiment	"G" level at blackout	Anxiety rating	Aggression rating
1	I	3·1	+++	0
	II	3·8	++	+++
2	I	3·6	+++	++
	II	4·0	+	++
3	I	3·9	++	+++
	II	3·8	+++	++
4	I	4·0	+	+++
	II	5·1	0	+++
5	I	3·3	++	0
	II	3·3	++	+

FIG. 3.—The subjects are identified by the numbers in the first column. The second column indicated the experiment and the third, fourth and fifth column the blackout level and psychiatric rating following each experiment.

following the second run. The other subject revealed a moderate increase in his *g*-tolerance in the second run in association with greatly reduced anxiety. The affect state of the third subject was judged to be essentially the same as in the first experiment and the *g*-tolerance also remained unchanged. The psychiatric rating and the *g*-tolerance of these subjects is listed on Fig. 3.

An interesting incident occurred at the laboratory which was not actually part of the experimental series but the results of which certainly are consistent with the findings. A student pilot had been sent to the laboratory for evaluation of his *g*-tolerance. The procedures usually carried out were a series of rides in the human centrifuge during a 3 or 4 day period when the subject's tolerance to centrifugal forces were evaluated. On the second day of his evaluation, the patient blacked out at 3·5 *g*. The examining physician on duty felt that the student pilot was in a state of acute panic following this ride and it appeared to him that the panic was in some way related to the student's blackout level since on a previous day this pilot had blacked-out at a considerably higher *g*. One of the authors was then called and told about the incident. The student was seen in the psychotherapeutic session lasting 1½ hr in which the causes for the anxiety were explored. An attempt was made to work through some of the sources of his anxiety and provoke a more aggressive state. Immediately following this interview, the subject was re-ridden on the centrifuge and his blackout level was re-established. During this series of

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rides, the subject was exposed to *g*-levels as high as 4·4 *g* but he did not blackout (COHEN *et al.*, 1957).

E. Catechol amine levels and variations in g-level

The experiments and observations described certainly suggest that emotions are related to *g*-tolerance and the level of catechol amines seen in the urine following exposure to positive acceleration. However, the presence of the relationship does not settle the question of causality. It is certainly conceivable that changes in subject's affect state could affect his response to centrifugation without postulating the release of catechol amines as a specific concomitant of the emotional state. Psychophysiologic factors might affect the vascular system perhaps through autonomic influences apart from any hormonal factors. Furthermore, blackout thresholds might be lowered in an acutely anxious person if his perceptual acuity decreased as he approached the panic level since he might be less able to perceive or respond to the panel of lights which was situated in front of him (used to assess his peripheral and central vision). The subject was expected to respond to the flashing on of these lights by pressing a button, indicating that he was still capable of responding to the lights. In cases where subjects were less able to perceive or respond, the change in catechol amines might be a function of the exposure to different *g*-levels rather than the result of affect changes, since subjects with decreased perception or response capability might merely be exposed to lower *g*-levels, hence, less vascular stress. It could be argued then, that the high noradrenaline levels seen in high-*g* subjects was due to the fact the subjects endured greater hypertensive stresses and that it was this factor that produced high noradrenaline levels. In other words, the noradrenaline level was the effect rather than the cause of the *g*-level reached.

Even if the emotional state of the person can influence the catechol amine excretion, the previous experiments did not clearly differentiate the relative role of vascular stress and emotional stress on the level of catechol amines seen in the urine. For this purpose, a series of experiments were undertaken in which vascular and emotional factors were focused on separately. The experiments which will be briefly mentioned in this section were primarily conducted by Dr. MCGOODALL at Duke University in conjunction with the personnel at the laboratory where the authors were working (GOODALL, in preparation). In these experiments, some of the subjects who participated in the previous centrifuge-affect experiments were used. The experiment was aimed at evaluating changes in catechol amine excretion ratios when subjects were exposed for a 1 hr period on different days to different intensity of *g*. For example, on different days, the subject was given a series of ten rides at 2 *g*, ten rides at 3 *g*, and ten rides at 4 *g*.

The results indicated that as the intensity of the vascular stress increased, the level of adrenaline and particularly noradrenaline rose. The ten rides at 4 *g* would seem to represent a far greater vascular stress than the ride profile used in the centrifuge-affect experiments. In those experiments in which attempts had been made to arouse the subjects emotionally the individuals also had ten rides, but the rides began at 3 "*g*" and progressively increased until blackout was reached. Subjects who participated in the particular series of experiments now being described all had blackout levels in the centrifuge-affect series about 4 *g*, in other words, the high-*g* subjects. Hence, they had no more than three rides over 4 *g* in the centrifuge

affect series. Although the vascular stress was less intense during the centrifuge-affect experiments the catechol amine levels during that series were much higher than those seen in the series described in this section. This would seem to indicate that the arousal of affective factors, although not the only one, is an important determinant of catechol amines released when subjects are riding the human centrifuge.

EXPERIMENT II

A. Response to mecholyl injections

In order to firmly establish the relationship of affect state, catechol amine levels and vascular responses, it appeared that the investigations had to be taken out of the framework of the centrifuge type of experiment because of the high degree of vascular stress which was imposed on the subjects. An experiment in which the imposed vascular stress was the same for each subject was obviously the next step. In addition, if this vascular stress could be minimal as well as constant for each subject then it would be much more apparent that catechol amine levels were more intimately related to the psychophysiologic state of the subject and not merely the degree of vascular stress to which they would be exposed. Further utilizing a totally different experimental situation would presumably result in different affective responses and would allow for the examination of catechol amine levels in the light of the differential affective response to two different stresses. Thus, another series of experiments was carried out using small doses of mecholyl (5 mg) as the physiologic stressor in order to answer some of the problems posed by the centrifuge experiment. It seemed desirable to use a drug with a hypotensive effect in subjects in a relatively immobile seated position so that the cardiovascular reflexes activated would be similar to those which were evoked on the centrifuge. Mecholyl, a methylated derivative of acetyl choline, was selected as the hypotensive agent. It was also felt that blood pressure determination would be a more objective endpoint than blackout.

The procedures followed were similar to those in the centrifuge experiment as described in detail in other publications (COHEN *et al.*, in press; ZUIDEMA *et al.*, 1957). The subjects urine was collected just prior to the experiment. They were then seated in a darkened, soundproof room and electrodes were placed for various bioelectric measures. A blood pressure cuff was placed on the subject's arm and attached outside the room to an automatic blood pressure pump. The pressure was recorded on oscillograph paper. The subjects were chastised by the experimenters immediately after the experiment began in order to provoke an affective response. After 20 min an injection of mecholyl was given subcutaneously and measures for 30 min or longer were carried out. Blood pressure was taken automatically (ZUIDEMA *et al.*, 1956a) every 30 sec, but the oscillographic record was not developed until the following day. Following the thirty minute post mecholyl period, a urine again was collected and the subjects were interviewed. The data of the three teams (psychiatric, vascular and neuro-hormonal) was collected and evaluated independently and compared only after they had been tabulated. Figure 4 shows the systolic blood pressure response of twelve subjects immediately following the injection of mecholyl and the psychiatric assessment after the experiment. All subjects judged to be primarily anxious gave hypotensive responses. Subject four was the only member of this group judged to be only moderately anxious in contrast to marked anxiety seen in subjects one, two, and

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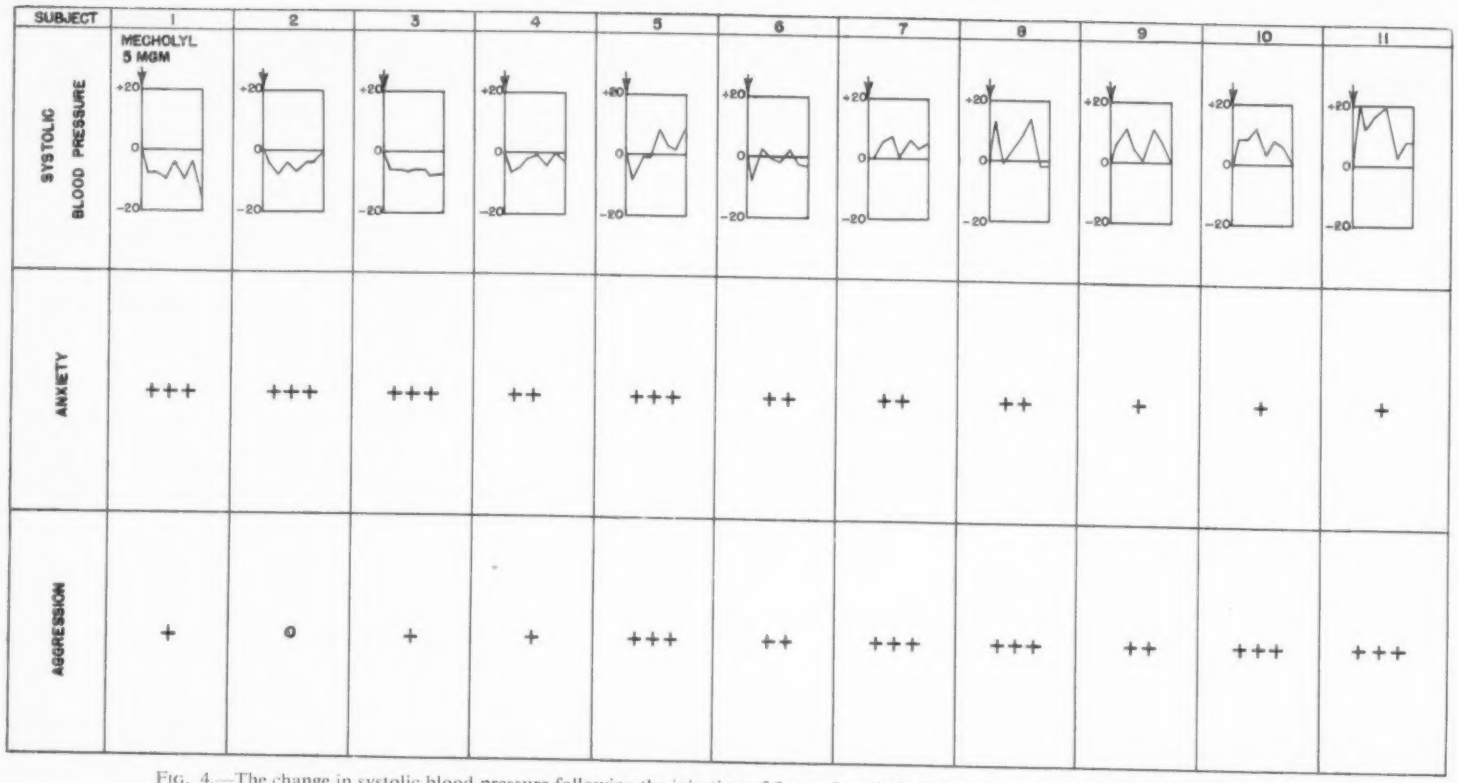

FIG. 4.—The change in systolic blood pressure following the injection of 5 mg of mecholyl is indicated in the upper horizontal column while the lower two columns contain the psychiatric ratings.

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three. He showed the least marked drop in blood pressure. It should be further noted that subjects one to four also had a drop in diastolic pressure which is not seen on this chart. Subjects five and six were evaluated as being anxious, but also appeared to demonstrate a sufficient quantity of aggressive ideas and thoughts so that neither state was designated as primary but the subjects were described as having equivalent amounts of anxiety and aggression. The subjects showed mixed or biphasic responses. Subjects five and six had decreases in diastolic pressure. However, in subject five, it increased progressively in the latter 15 min. Subjects seven to twelve were all felt to have been primarily aggressive and all demonstrated hypertensive responses. Subjects seven, eight and eleven had an increase in diastolic pressure. Subject seven had a mixed response and subject ten showed a slight decrease. Figure 5 lists the catechol

Subject	Pre-run		Post-run		Post-run Psychiatric evaluation	
	Adrenaline mg /hr	Noradrenaline	Adrenaline	Noradrenaline	Anxiety	Aggression
1	—	—	2·1	2·4	+++	+
2	0·89	2·92	2·45	1·89	+++	0
3	1·82	1·92	0·43	1·72	+++	+
4	0·53	1·83	1·19	2·17	++	+
6	1·78	2·55	2·90	4·30	++	++
7	0·46	4·77	2·22	5·01	++	+++
8	0·62	2·23	0·96	8·0	++	+++
10	0·46	2·08	0·25	2·75	+	+++

FIG. 5.—The pre- and post-run adrenaline and noradrenaline levels of some of the subjects in the mecholyl experiment is listed in the first four columns while the last two columns contain the psychiatric ratings.

amine excretory rates and it can be noted that subject ten's ratios are consistent with the psychiatric evaluation (primary state of aggression with low level anxiety). However, the level of noradrenaline is lower than any of the others in the aggressive, hypertensive group and may not have been sufficient to maintain vasoconstrictive tone and peripheral resistance against the vasodilatory action of mecholyl. This figure also lists eight of the subjects who were shown in the previous chart who had catechol amine levels taken before and after the experiment. Subject two, who was felt to have been in a near panic state following the experiment (and had a hypotensive vascular response), had a very unusual change in his catechol amines with a marked increase in adrenaline and a decrease in noradrenaline so that his post-run adrenaline was higher than the noradrenaline in spite of the fact that his pre-run noradrenaline was considerably higher. The post-run noradrenaline levels in the anxious subjects (one to four), who gave hypotensive vascular responses, were lower than the level of the subject who had a mixed vascular response (six) and a hypertensive vascular response (seven, eight and ten). The results strongly suggested the affect state, catechol amine levels and vascular responses were interrelated. Naturally all the subjects did not fit precise patterns for many reasons. It is obvious that vascular responses are not only dependent or related to catechol amine levels. Further, it seems plausible that the relationship between emotional states and vascular responses involves other humoral and neural factors in addition to the catechol amines. Thirdly, the psychiatric assessment, although it attempted to quantitate, could actually be depended upon for no more than a qualitative assessment of the primary affect state.

Bioelectric measures used during this experiment to assess the level of arousal (EEG and GSR) indicated that subjects three and ten were less aroused than the psychiatric evaluation indicated. All other subjects showed a most striking correlation in the various parameters. Subject ten's findings were mentioned previously. Subject three showed a drop in adrenaline and noradrenaline in the post-run specimen. His bioelectric records indicated he was less aroused during the experiment than before it, when he had become quite anxious over some incidents occurring in the laboratory. His pre-run adrenaline is the highest in the series. His affect state during the experiment was felt to be primarily anxious but of a low level intensity, after the bioelectric records were reviewed (the bioelectric data will be presented in a subsequent paper). The hypotensive response in this subject was felt to be primarily a function of insufficient noradrenaline to maintain vascular tone in face of the vasodilatory factors (mecholyl, posture and immobility), although the level may have been sufficient to maintain his vascular tone during a resting period without any extraneous vascular stress.

B. Altered affect state and exposure to mecholyl

The question of causal relationship can again be raised, that is, what is the meaning of the inter-individual differences which are seen. Do these represent differences in innate physiological and hormonal response characteristics or are they truly the result of different affect states being aroused in the various subjects. If the former case were true, that is innate physiologic and hormonal patterns, then the affective constellations noted might possibly be a function of such factors as the level of catechol amines on thinking and behaviour patterns. For example, persons with high noradrenaline/adrenaline secretory ratios might be aggressive and nonanxious because of some effect on behaviour by the excess or lack of the neurohormones. This would, of course, be somewhat in contrast to the hypothesis being primarily postulated. Because of this and for other reasons, it was felt essential to demonstrate that intra-individual variations in affect state produced changes in vascular responses. In this way then, it could be shown that the relationships noted were not merely due to inter-individual response characteristics, but truly a function of the arousal of the specific affects.

The aim in this second series of mecholyl experiments was to produce specific alterations of affect state. It was felt that no specific stimuli could be chosen which would necessarily change all the subjects' affect states in the desired directions. However, all the subjects were persons working at the laboratory and this was advantageous in that a great deal of personal contact and hence, information about the subjects was possessed. That is their psychologic, social and work characteristics were known to the experimenters. Because of this, it was decided that attempts would be made to alter the affect state by creating situations just prior to the experiment which were specifically designed for each subject, and aimed at producing a specific emotional state. Nevertheless, the only logical approach which could be taken was to avoid making an initial assumption of the affect state and leave the final decision up to the post-run interview evaluation. The interviewer would make every attempt to cover the same topics as in the interview conducted after the first mecholyl experiment, and then compare the quantity and quality of the affect aroused in the two interviews on the basis of the behaviour, the verbalizations and the associations.

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For example, one subject (four) during the first mecholyl experiment had displayed moderate anxiety and minimal aggression and a hypotensive response to mecholyl. He had shown fairly marked increase in adrenaline with a very slight increase in noradrenaline. The subjects' anxiety in the first experiment appeared to be related to a large extent to a lack of knowledge of the pharmacology of the drug being used. It was known that this person was in general an aggressive person who felt most comfortable when he could control and master situations. Situations in which he lacked control, particularly if this was a function of a lack of adequate information, were anxiety provoking for him. Furthermore, this person was a meticulous compulsive worker who had little tolerance for inefficiency in himself and in others.

With these characteristics in mind a plan to alter his affect state prior to the experiment was formulated. Before the subject came to the experimental room, one of the investigators conferred with him and discussed in detail the profile of the experiment, the pharmacologic effects of the drug and the physiologic reactions which he could anticipate. It was thought that by understanding the factors with which he was unfamiliar in the experiment, there would be a reduction in his anxiety by allowing him to feel that he could master the situation and thus reducing the threat of the unknown. In an attempt to arouse some anger and permit him to discharge this in an acceptable manner, the technicians were instructed to engage in activities during the period the electrodes were being placed which were calculated to annoy and provoke him. For example, they were to carry out their tasks in an inefficient manner so that it resulted in some personal discomfort to the subject.

A series of events occurred while he was being prepared for the experiment in which the inefficiency and apparent lack of care of the technicians resulted in the subject becoming 'messed up' and with this he verbalized discomfort and annoyance with the way in which the situation was being handled.

In the interview which followed the experiment minimal anxiety was present regarding the effects of the drug, much less than that expressed after the first run. He presented a picture of being in much better control and being able to master the experimental stresses more comfortably and effectively. His anger was judged to be moderate and was illustrated when he said that during the experiment he had the impulse to defaecate. The interviewer wondered whether this could have been related to any of his feelings during the experiment. The subject laughed and said: "Well, I might well have felt like defaecating on the whole mess of you." It was suggested that the subject was aroused to anger but was able to handle the effect engendered. The affect evaluation then, during the second experiment, was just the reverse of the first.

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Figure 6 shows the change in systolic and diastolic pressure in the subjects tested on two occasions. Note that subject four in the first experiment was judged 2 plus anxiety, 1 plus aggression and as stated before he gave a hypotensive systolic and diastolic response to mecholyl. But on the second run where the subject was judged 2 plus aggression and 1 plus anxiety, there is a shift in his blood pressure pattern following mecholyl, with a hypertensive systolic and diastolic response.

Four subjects in all were treated in a manner similar to that just described. Two subjects who had been anxious had given hypotensive responses (three and four) and two subjects who had given hypertensive responses and had been evaluated as primarily aggressive (seven and eleven) were chosen. In each case an attempt was made to reverse their affective response. The anxious subjects were judged to have been primarily aggressive the second time and their blood pressure curves were reversed. The aggressive subjects although not felt by the interviewers to have been completely reversed affect-wise did manifest higher levels of anxiety and lower levels of aggression. Their vascular responses were also changed appropriately. Unfortunately, the urine specimens from the experiments were lost in the mailing process, therefore only the affect and the vascular evaluation is charted.

C. Variations in response to different 'stress' situations

A further illustration of the fact that cardiovascular responses are directly influenced by emotional factors is seen when one compares the responses of some

subjects to mecholyl and their response during the centrifuge experiment. Although the cardiovascular stressors in the two experiments differ in magnitude, it might be assumed that high-*g* persons would give hypertensive responses to mecholyl because of an active carotid sinus mechanism and/or high endogenous noradrenaline levels. On the other hand, if affective factors are important in determining the cardiovascular responses of persons then the above would not necessarily hold true. The setting of

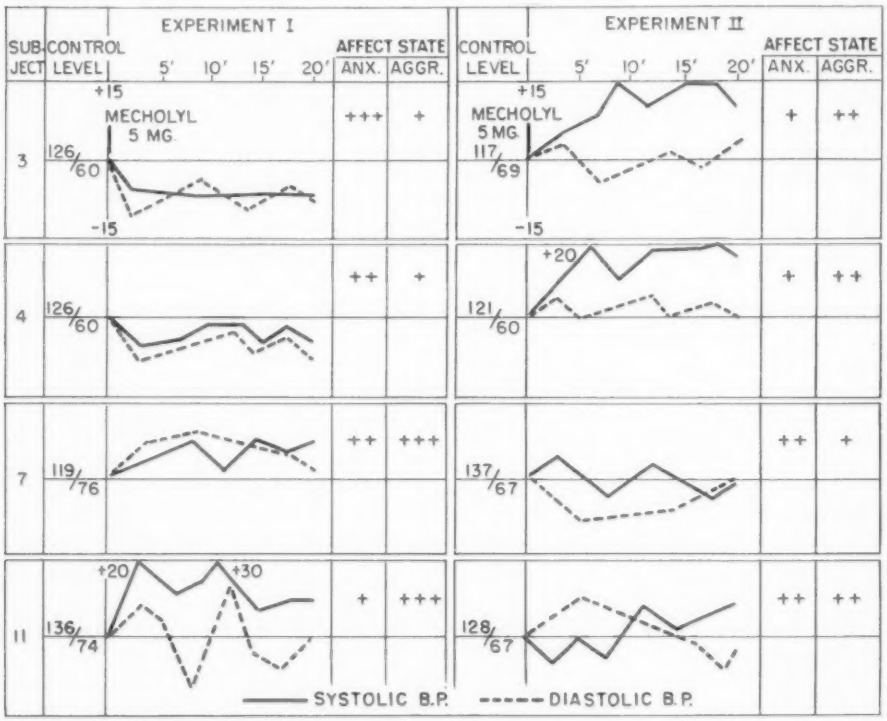

FIG. 6.—The systolic (solid line) and diastolic (broken line) blood pressure change following the injection of mecholyl during two separate experiments (on right and left side of double vertical centre line) is indicated. The affect state associated with each vascular response is also charted.

The premecholyl control blood pressure is charted to the left of the lines indicating the change following mecholyl. The central horizontal line in each patient's record signifies the control level.

The scale provides for a deviation of plus or minus 15 mm of mercury. In the cases where the change exceeded this it is noted (subject four, experiment II; subject two, experiment I.)

the two experiments was quite different and might lead to the arousal of the different feelings. If this were not an important factor then the only difference subjects would show might well be in the magnitude of response because of the varying intensity of the cardiovascular stress in the two experiments.

When one compares the mecholyl and the centrifuge experiments, there are certain obvious differences in terms of the meaning the situations could have for different subjects. The mecholyl experiment is characterized by the subject being isolated in a darkened room and kept in a relatively immobile state. The discomfort he may feel primarily emanates from internal sources even though external forces

may contribute to it. For example, the mecholyl injection may be the cause of various physical sensations. But when the sensation occurs the subject is alone and no external reason for the sensation is present. In general, during the mecholyl test, there are very few external stimuli.

The centrifuge experiment on the other hand exposes the subject to a marked external stressor to which the physical sensations can be directly attributed. Although

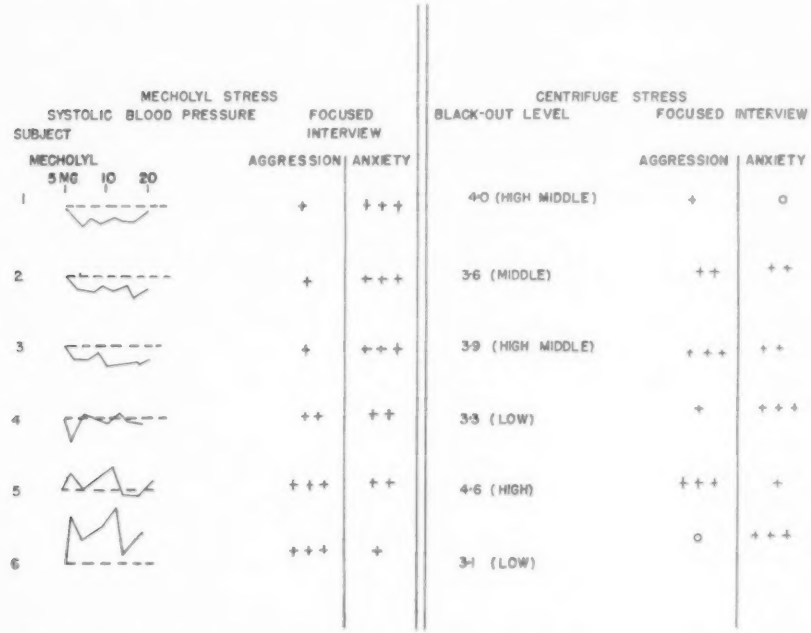

FIG. 7.—The left side of the chart indicates the systolic blood pressure change following an injection of mecholyl and the psychiatric assessment associated with this. The right side of the chart indicates the blackout level and affect state of the same subjects on different occasions.

the subject was asked to do a minimal amount of moving during the centrifugation, the subject did have to push buttons to respond to the light panel and hence was engaged in some activity. Although isolated in the cab, he does speak to the operators periodically since the symptoms must be determined after each ride.

These two situations could be approached with an entirely different attitude. The association to the centrifuge ride may be quite different than those to the isolation, immobility and darkness during the mecholyl experiment.

Figure 7 demonstrates the responses of subjects who participated in both series of experiments. The systolic blood pressure change after the injection of mecholyl are shown together with the interview evaluation which followed. In addition the subject's *g*-tolerance and affect evaluation following the centrifuge experiment is charted.

Subjects one, two and three were all judged to have been markedly anxious (3 plus) during the mecholyl experiment and they all demonstrated hypotensive responses. When these subjects were tested on the centrifuge they varied somewhat

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in their affective response but in no case was their anxiety level considered as high as during the mecholyl experiment. It is significant that none of these subjects showed low *g*-tolerance. The second subject shown on the chart was felt to have been aroused to moderate anxiety and moderate aggression in contrast to the first and third subjects who were evaluated as being primarily aggressive. Subject two had the lowest tolerance in this group which correlated with the difference in his affect state evaluation. The fourth subject shown in the chart was evaluated as having mixed affect state during mecholyl test; moderate aggression and anxiety. However, on the centrifuge he was apparently aroused to marked anxiety and blacked out at a fairly low-*g*, indicating poor vascular reactivity to this type of hypotensive stress. Subjects five and six were both judged to have demonstrated marked aggression during the mecholyl and both had hypertensive vascular responses. The sixth subject appeared to be less anxious than the fifth and this subject's blood pressure increase was more marked. In the centrifuge, these subjects had variable responses. The fifth subject continued to show marked aggression and his anxiety level was less than during the mecholyl test. The subjects' *g*-tolerance was extremely high and indicated that the vascular compensatory activity to the hypotensive centrifugal stress was quite marked. The sixth subject was in a near panic state during the centrifuge run and blacked-out at a very low level. This indicated a very poor compensation to the vascular stress which was in sharp contrast to his active vascular response to mecholyl. In all the cases in the series, the affective state of the subjects correlated with the physiologic response. High levels of anxiety were associated with poor vascular reactivity and low levels of anxiety and high degrees of aggression were associated with active cardiovascular reflex activity. The content of the subjects interviews revealed that much of the subjects emotional reactions during the experiments was determined by feelings related to the meaning of the experimental situation. These cases suggested very strongly that the subjects emotional state plays an extremely important role in contributing to the vascular response patterns. It is also quite clear that it is essential to evaluate the meaning of the stresses in an experimental situation to the individual subjects in order to adequately understand their response.

D. Psycho-physiologic response tendency profiles

It might be thought that the object of the experimental procedures was to obviate the importance of inter-individual differences and highlight the influence of the immediate psychophysiologic state in producing responses to cardiovascular stress. It is true the results implicate the extremely important role of affective factors in governing vascular responses at any moment in time. However, the importance of the general response tendencies of an individual cannot be overlooked.

One major difficulty in approaching this area is answering the question: "What is the most important factor in the formation of ones physiologic and psychologic response patterns?" The age old controversy of hereditary and environmental influences can be brought up at this point. Two rather extreme approaches could be taken. One could hold that a person is born with certain innate biochemical, hormonal, physiologic and behavioural characteristics which manifest themselves at specific times, dependent on their genetic structure. Even with this rigid approach, questions still arise. For example, the insolvable riddle of cause and effect, arises when one asks if all the characteristics of the person, e.g. behavioural, hormonal

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levels, etc., are inborn or is the hormonal level inborn and the cause of the behaviour pattern.

The second extreme approach which could be taken is that all traits are acquired and the inter-relationship of hormonal, psychologic, physiologic factors is due to the setting up of circuits in the central nervous system as the result of experiences which link the various responses.

The solution to the above enigma will not be attempted in this report. The only logical contention which can be held by the authors is that there is some validity to each of the above approaches with both sets of factors contributing to the ultimate response tendencies of individuals.

In the centrifuge and mecholyl experiments, we mainly considered the correlation of psychologic and physiologic data from experiments performed on a specific day under a specific set of conditions. If the focus is changed then one could consider the average reaction tendencies and determine what kind of relationships exist between profiles of subjects psychologic, biochemical and hormonal characteristics and average physiologic responsivity (average rather than response on any one day). Unfortunately, the only average physiologic responses which can be presented are the average *g*-tolerances. That is the *g*-tolerance of subjects over a period of time (1 year) rather than the *g*-tolerance of any specific day. These tolerances were not obtained during experimental procedures as outlined in this report. They were obtained during a variety of experiments which were conducted at this laboratory. In none of these experiments were attempts made to emotionally arouse the subject or evaluate his affect state. Of course, it is probable that subjects were aroused affectively either before or during the runs for reasons either attributable to their life situation or the centrifuge itself. (A series of non-emotionally arousing mecholyl tests is planned and it is anticipated the results would correlate well with average *g*-tolerance since both stresses might reflect the capability of the cardiovascular system to compensate to a hypotensive stimulus.)

On Fig. 8 are the profiles of six subjects whose *g*-tolerance was relatively consistent and did not vary more than 0·4 *g* during the year. Three high-*g* (4·2) and three low-*g* (less than 3·7) subjects are included. Four parameters are compared: (1) FTT scores; (2) average pre-experimental noradrenaline; (3) average pre-experimental Adrenaline levels; (4) hippuric acid level.

The FTT is believed to be an indicator of the aggressive tendencies of the subject. The higher the score, the greater the subjects aggressive expressions in the test. This is interpreted to mean, that the subject is more liable to react to life situations aggressively than persons with a low score. The lower the score the less aggressive the subject is taken to be and the greater his proneness to anxiety. However, as has already been pointed out, the test's primary value appears to be in picking up aggressive tendencies and is only of secondary value in determining anxiety proneness.

The noradrenaline and adrenaline levels are the average of pre-run specimens from a series of experiments in which the subject was a participant. This might be taken to reflect the level of catechol amines when the subject is anticipating a stressful situation (in this case an experiment). On the other hand, they may represent his endogenous resting level. Twenty-four hour levels or waking specimens are probably more desirable for a comparison such as this and will be obtained in the future.

The hippuric acid level is used as an indicator of the degree of free floating

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chronic anxiety. Of course, it does not reflect the degree of anxiety that can be anticipated in any particular experimental situation, but may be related to the subject's chronic free floating anxiety level.

All the parameters are charted in terms of the percentage of average levels or average score, e.g. the average normal hippuric acid excretion level, the average normal catechol amine levels of persons engaged in moderate activity (VON EULER) and the average score of the total group tested with the FTT.

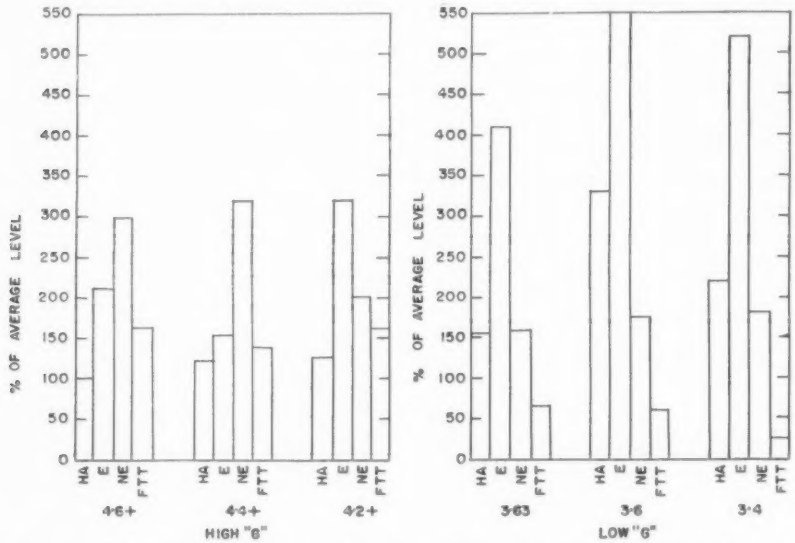

FIG. 8.—This figure demonstrates a profile of three high and three low-*g* subjects. Hippuric acid excretion rate, pre-stress levels of adrenaline and noradrenaline and FTT score are expressed in percentage of normal or average level seen.

The catechol amine levels were obtained in at least two separate experiments and in some cases as many as six experiments in which the subject was a participant. Of course, the subjects pre-run control levels did vary particularly when he was anticipating different experiments (e.g. a mecholyl or centrifuge pre-run control). However, the figures for any one subject were fairly constant. It should be pointed out that the authors are fully cognizant of the inadequacy of the measure being used and it is merely being presented as suggestive evidence. Further work is contemplated in which average catechol amine levels will be collected in a more meaningful and statistically reliable fashion.

The chart of the subjects results reveals rather clear differences between the high and low *g*-subjects. The high-*g* subjects have lower hippuric acid excretion rates and adrenaline levels and higher noradrenaline levels and FTT scores. There is no exception in any parameter in any of the subjects in the two groups.

The results then certainly suggest that the relationships noted previously in experiments focusing on the psychophysiologic state of persons at a specific moment in time are also seen when specific aspects of the subjects usual psychophysiologic responses are examined. The results could be taken to mean that subjects who demonstrate a high degree of aggressivity on the FTT and hence, probably in life

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situations (at least those depicted on the FTT) and who have low levels of chronic free floating anxiety (hippuric acid), are persons who have higher 'non-stress' (or still better pre-stress) levels of noradrenaline and lower levels of adrenaline. Furthermore, over a period of time these persons consistently demonstrate a greater ability to withstand the vascular stress of the human centrifuge (perhaps it is wiser to merely say the stress of the human centrifuge rather than limiting the tolerance on the human centrifuge to vascular factors only).

It would appear that to some extent, some of the subjects can be grouped on the basis of the FTT into two groups: (1) Overtly aggressive, autonomous, somewhat impulsive, active individuals who demonstrate little anxiety with certain aggression provoking situations and in general, have low levels of free floating anxiety. (2) Others appear to be chronically anxious or fearful persons who show traits of passivity, dependency and submission. In certain situations they are fearful of being aggressed against and unable to tolerate aggressive impulses of their own.

Subjects who clearly fell into one of these two psychologic groupings appeared to be distinguishable on the basis of their average response to *g*-stress over a period of time. Although there was some day-to-day variation in their *g*-tolerance, it was within a limited *g*-range (low or high).

Therefore, the approach to the study of psychophysiologic relationships would appear to require much more than merely relating changes in several parameters during exposure to a stress of a certain type which affects a specific system. It appears to necessitate the assessment of the psychophysiologic response characteristics of the subject immediately preceding the imposition of the stress, the usual response pattern of the subject to the specific stress situation and the overall responses patterns of the subject to life situations.

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DISCUSSION

The findings of the experiments which have been described imply that catechol amine levels are important correlates of an individuals vascular response. Hypotensive responses to injections of mecholyl and exposure to centrifugal stress, manifested by low *g*-tolerance, occur in conjunction with low excretory levels of urinary noradrenaline and moderate or high levels of urinary adrenaline. On the other hand, hypotensive responses to mecholyl and low tolerances to centrifugal stress is accompanied by low noradrenaline levels and increased adrenaline.

It certainly was also strongly suggested that the type and intensity of affect present during the experimental situation can affect the vascular response. The effect of affective arousal on vascular responsivity appears to be mediated to a large extent by the differential release of adrenaline/noradrenaline, at least as noted in urinary excretory levels. Hypotensive vascular responses, together with low levels of noradrenaline and moderate to high levels of adrenaline occurred in subjects who were judged to be markedly anxious and demonstrated minimal aggressive feelings and behaviour. However, subjects with hypertensive responses and high levels of noradrenaline demonstrated marked aggressive feelings and minimal to moderate anxiety. Observations by other investigators tend to support the relationship being proposed here. ELMADJIAN (1957) examined the urine of the normal person and psychiatric patients for the level of adrenaline and noradrenaline. Aggressive hostile patients had increased excretion of noradrenaline and the fearful one increased excretions of adrenaline. Furthermore, he found that when subjects were given

mecholyl, the greater the hypotensive response the lower was the noradrenaline level. He further noted that the infusion of adrenaline and noradrenaline during the mecholyl test caused an increase in hypotensive response with adrenaline and a decrease with noradrenaline.

It does appear then that specific emotional states are associated with variations in catechol amine levels and vascular responses. This may be the result of the activating of the specific hypothalamic areas, for example, fear and rage and the selective release of these neurohormones. It has already been demonstrated that specific areas exist in the hypothalamus which when stimulated cause a selective release of adrenaline and noradrenaline (FOLKOW and VON EULER, 1954). Furthermore, there is evidence that cells in the adrenal medulla may release adrenaline and noradrenaline independently and these cells may be innervated by different hypothalamic areas (HILLARP, 1953). The effect of the arousal of affects on vascular responses is obviously more complicated than an–affect–hypothalamic–hormonal relationship. When one considers the number of central nervous system areas which can lead to vasomotor changes this certainly becomes clear. Furthermore, the central nervous system mechanisms of thinking, feeling and behaviour are far from being clearly delineated.

On the basis of the work which has been presented, the evidence for the release of adrenaline in response to anxiety is much clearer than the relationship of anger and aggression to noradrenaline. It may be that it is not only the affect which is of importance but such factors as muscular tension associated with aggressive activities which are related to noradrenaline release. VON EULER and others state that any kind of muscular activity causing a widening of the vascular bed in the muscles is compensated for by blood pressure homeostatic mechanisms which cause vasoconstriction (which is associated with noradrenaline release) is in part responsible for the association of the affect of anger or aggressive impulses with an increase in noradrenaline. However, it is also true that high levels of anxiety could lead to increased muscular activity and motoric responses, although these might be directed at flight or escape rather than fight or attack. Therefore it would seem plausible to assume that there are many instances in which it is not the difference in quantity of activity that causes variations in hormone levels with different affects. It is suggested that perhaps there is a qualitative factor involved also. Whether this is based on two different central mechanisms for each affect, e.g. fear and anger, and the different patterns or muscular activity aroused by the two affects, is still a moot question.

The relationship of specific affective constellations and catechol amine levels may have its basis on the association of certain infantile emotional responses and their physiological concomitants. An infant's reaction to unpleasant inner sensations associated with deprivation, frustration or pain can be perhaps characterized as a massive dedifferentiated discharge phenomena. *A propros* of this is WESTS' (1951) study which showed that neonates have high levels of noradrenaline and low levels of adrenaline. The high noradrenaline levels may in part be the result of this diffuse muscular activity. As infants develop their first attempts to cope with their increased tension may be defined as primitive aggression. Hence, it is postulated that the affect of rage or anger or the carrying out of aggressive behaviour might re-excite these circuits and lead to the release of noradrenaline even in the absence of the diffuse muscular activity which was the original stimulus calling forth the noradrenaline

release. As muscular coordination increases, this ability might be used in the service of goal directed activity. The amount of activity might become considerably less than the diffuse uncoordinated activity of the infant, but the association of more specific aggressive behaviour and feelings with the original primitive reaction patterns may lead to the reexcitement of the original circuits. Although the manifest behaviour may be culturally determined modification of aggressive behaviour, the circuits associated with primitive rage may be aroused. Thus, the hormonal reaction may be similar to that of the infant in a primitive rage state although the affect perceived is far less intense, and the muscular activity less diffuse.

Man's long growing-up process subjects him to a multiplicity of experiences which must be considered if one is to understand a person's response to stress, be it experimental or life. A person's background and experiences to a large extent account for those stimuli which are regarded as stressful as well as the relative intensity of his reaction to stress. Hence, extreme differences may be noted between individuals and their reactivity to psychologic or physiologic stimuli. It is quite clear then that the response to stress depends on the type of individual we are dealing with as determined by his genetics and the dynamic facts of his background. Then add to this mass of variables, the problem of the state of the organism at the time a stressor impinges. Without belabouring the point, suffice it to say that the meaning of the stress to an individual as well as his physiologic response to it will depend on a persons' psychophysiologic state at any particular moment in time. It is clear then, that one must identify the meaning of a specific stress for that one individual at a specific time in order really adequately to assess man's responsivity. It would seem that only by identifying the specific dynamic, affective, neurophysiologic, biochemical, hormonal and physiologic components of a response will one be able to make definitive statements regarding the total psychophysiologic mechanisms operative in any life or experimental stress situation.

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SUMMARY

The arousal of specific emotional states such as anxiety and anger together with the activation of specific behavioural modes associated with aggressive feelings and ideas is hypothesized to be related to characteristic vascular responses, and may be a reflection of selective release of adrenaline and noradrenaline.

Projective tests used in a focused manner to evaluate specific emotional traits indicated that the characteristic aggressive behavioural traits of a person and the level of chronic anxiety, as reflected in the hippuric acid excretory rate, were associated with the average compensatory response characteristics of the subjects to a hypotensive stressor (the human centrifuge).

Vascular compensatory activity in response to human centrifugation as well as to small doses of mecholyl appeared to be related to the affective and behavioural response of the subjects. Anxious, non-aggressive responses were associated less vascular compensatory responses than those seen in subjects who demonstrated aggressive responses and a low level of anxiety. The aggressive subjects had higher noradrenaline and lower adrenaline urinary levels than the anxious subjects. When the subjects were exposed to a second experiment in which their affective and behavioural response was altered, the vascular response and catechol amine ratio was changed. The shift in the physiologic and hormonal response in the second experiment was consistent with the correlation demonstrated in the first experiments.

Hence, intra- and inter-individual variations in vascular responses and catechol amine levels were related to specific emotional states and behavioural responses.

The possible mechanism mediating the psycho–physiological chain of events is discussed with consideration of psychodynamic, neurophysiologic, hormonal and vascular factors.

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THE HIPPURIC ACID INDEX: APPLICATION OF THE HIPPURIC ACID TEST AS AN INDEX OF HUMAN FREE ANXIETY

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THE purpose of this paper is to present evidence suggesting that the hippuric acid test may be applied as an objective and quantitative measure of free anxiety in psychiatric patients. Evidence is presented that refinements may considerably increase the accuracy of the measurement. The term "hippuric acid index" is used here to designate these refinements as now formulated.

BACKGROUND OF THE TEST

The main points of clinical and experimental background bearing upon the psychiatric application of the hippuric acid test may be summarized as follows:

General factors. (1) Introduced by QUICK (1933) as a measure of liver function, the hippuric acid synthesis test has had wide clinical use for this purpose (QUICK, 1933). It consists of oral or intravenous administration of a loading dose of sodium benzoate with collection and analysis of the urine voided after a timed interval. Most of the administered benzoate is conjugated (detoxified) with endogenous glycine, the simplest amino acid, to form hippurate. The latter is excreted rapidly in the urine and can be measured as hippuric acid in grammes.

Liver disease ordinarily impairs the synthesis of hippurate from benzoate and gives low values on the test (LICHTMAN, 1953). More rarely, mild but clinically evident liver disease apparently accelerates the synthesis and gives higher than normal values on the test (ROSENBERG and SOSKIN, 1941). Partly because of this biphasic aspect, the test has decreased in usefulness as a measure of liver function.

Urinary tract disease impairs the excretion of hippuric acid (KOHLSTAEDT and HELMER, 1936). Prostatism lowers the excretion by the amount of residual urine, unless catheterization is done, and kidney disease sufficient to impair urea clearance invalidates the test.

(2) Catatonic schizophrenia, hyperthyroidism, and a variety of debilitating conditions without associated liver disease have been demonstrated repeatedly to give abnormally low values on the hippuric acid test. (QUASTEL and WALES, 1938; WONG, 1945; GRAETZ *et al.*, 1954; BARTELS, 1938; BOYCE and MCFETRIDGE, 1938; MUSSER *et al.*, 1955; PAULSON and WYLER, 1942; SHERLOCK, 1946; PROBSTEIN and LONDE, 1942; STERN *et al.*, 1946). Test values in such conditions generally return to normal if glycine is administered before the injection of sodium benzoate.

Measurements of the blood concentrations of glycine or of its metabolic precursors, however, are not adequate to predict the amount of hippuric acid excretion (DE VRIES and ALEXANDER, 1948; BASOWITZ *et al.*, 1955). Available evidence suggests that the limiting factor in such conditions may be the size of the stores of available glycine and its precursors in tissues and body fluids.

(3) There is evidence that hippuric acid excretion fluctuates predictably in healthy persons with the normal ovarian cycle, being less at the time of menstruation than in the premenstrual period (HEILIG and KANTIENGAR, 1942; FREED and KROGER, 1950). Other clinical evidence suggests that the hippuric acid test is influenced by the activity of the pituitary-adrenal cortical axis (THORN *et al.*, 1942; SELYE and FORTIER, 1950; GRAETZ *et al.*, 1954).

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Psychiatric studies. (1) PERSKY, GRINKER, MIRSKY and GAMM (1949) restudied the hippuric acid test on the basis of earlier observations and reintroduced it as a tool uniquely applicable to psychiatric research (PERSKY *et al.*, 1949, 1950). In patients with neurotic free anxiety, the test gave abnormally high values, with the quantity of hyperexcretion of hippuric acid being roughly proportional to the intensity of the free anxiety. Catatonic schizophrenia gave low values on the test, the amount of hypoexcretion being roughly proportional to the degree of catatonia (PERSKY *et al.*, 1952). With successful psychiatric therapy, regardless of type, these test values returned to normal.

In case studies of psychiatric patients subjected to repeated testing, individual fluctuations in the intensity of free anxiety or in the degree of catatonia were associated with appropriate parallel changes in hippuric acid test values with a high degree of significance. It was postulated that catatonic schizophrenia and free anxiety define a gradient of psychomotor activity and that hippuric acid excretion is a quantitative correlate of an individual's position on this gradient (PERSKY *et al.*, 1952).

(2) In healthy persons, the hippuric acid test ordinarily gives reproducible results with a modest range of error on repeated testing. Test values demonstrate stability both with the passage of time and when the individual is subjected to transitory stress situations (PERSKY *et al.*, 1949, 1950, 1952; ZIEVE and HANSON, 1953; STERN *et al.*, 1946). Various stress techniques, such as college examinations and the hypnotic induction of anxiety, failed to elevate test values. Except for autonomic drugs, some of which decreased hippuric acid excretion, drugs did not change test values unless abreaction profoundly altered the level of free anxiety. In the latter cases, rapid increases in test values were found.

Studies of healthy young men in paratroop training have documented the uniqueness of the hippuric acid test as a biochemical index of stress response (PERSKY, 1953; BASOWITZ *et al.*, 1954, 1955). Unlike other metabolic indices which tend to change in response to less intense stress situations, hippuric acid excretion appears to rise significantly only as stress approaches severely pathologic levels, paralleling the development of clinically meaningful free anxiety.

(3) *p*-Aminohippuric acid, chemically analogous to hippuric acid, has been shown to exhibit analogous properties for psychiatric application (MUSSER *et al.*, 1955). MUSSER, DEISS and LORENZ (1955) have been able to differentiate a group of patients with chronic free anxiety from a group of known hyperthyroid patients on the basis of hyperexcretion and hypoexcretion of this substance, respectively.

Physical variables. (1) At least two investigations have demonstrated significant positive correlations between the normal amount of hippuric acid excretion and body size (HEPLER and GURLEY, 1942; SCURRY and FIELD, 1943). SCURRY and FIELD (1943) have determined formulas to predict individual normal values for the intravenous hippuric acid test, based on body weight and on surface area.

(2) There is adequate evidence that the normal value for the hippuric acid test decreases with increasing age (RAFSKY and NEWMAN, 1943; PAULSON and WYLER, 1942; STERN *et al.*, 1945, 1946; SCURRY and FIELD, 1943; PINCUS, 1950). Although especially noted in the senile age group, the evidence suggests a continuous inverse relationship between age and hippuric acid excretion throughout the adult life span.

(3) Because the measurement of synthesized hippuric acid depends upon its excretion in the urine, a low volume of urine during the test interval might be expected to decrease the normal value for the hippuric acid test. The evidence from several studies, however, is contradictory, and the use of different methods makes comparisons uncertain (MACHELLA *et al.*, 1942; SHERLOCK, 1946; SCHEINBERG and MYERS, 1948; MARRON, 1941; HEPLER and GURLEY, 1942; QUICK, 1939; PERSKY *et al.*, 1950). It may tentatively be concluded that at least a minimum urine volume is required for the test to be valid.

CONCEPT OF FREE ANXIETY

Free anxiety (overt neurotic anxiety) is a subjective feeling of heightened tension and diffuse uneasiness defined as "the conscious and reportable experience of intense dread and foreboding, conceptualized as internally derived and unrelated to external threat (PERSKY *et al.*, 1949, 1950, 1952; BASOWITZ *et al.*, 1955; MUSSER *et al.*, 1955; NOYES, 1953). It is not merely fear because it lacks a specific object. It is a painful, irrational dread of situations which covertly symbolize unconscious conflicts and

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impulses. Present knowledge places free anxiety in a central position in the dynamics of human behaviour as the essential and characteristic factor in neurosis.

Free anxiety is clinically quantifiable in terms of degree, intensity, or severity (PERSKY *et al.*, 1949; BASOWITZ *et al.*, 1955; BUSS *et al.*, 1955; DAVIDS, 1955). Its clinical assessment must depend primarily upon the subject's accurate report. Empirically, certain characteristic concomitants of free anxiety may prove to be useful supplements in evaluating the subject's report but do not in themselves define free anxiety. These concomitants, including the conventional "signs and symptoms" of anxiety, are conceived merely as correlates, reactions associated to varying degrees with free anxiety. In this sense, laboratory indices, such as the hippuric acid test, may be measures of certain biochemical concomitants of free anxiety.

PRESENT STUDY

In view of the complex background of the hippuric acid test, and in order to evaluate the interesting possibility that the excretion of this substance might prove applicable as a diagnostic test for "anxiety neurosis," we undertook a pilot study designed to shed light on the following questions:

(1) Can trained psychiatric observers independently rate the clinical level of free anxiety of subjects with enough agreement to establish confidence in the rating as a standard of comparison for other measures? (PERSKY *et al.*, 1952; BASOWITZ *et al.*, 1955; BUSS *et al.*, 1955; DAVIDS, 1955).

(2) When subjects are clinically rated along the gradient of level of free anxiety, with what accuracy does hippuric acid excretion predict an individual's position upon this gradient? (PERSKY *et al.*, 1952; BASOWITZ *et al.*, 1955).

(3) Are hyperthyroid patients, clinically rated for level of free anxiety, differentiated from similarly rated non-hyperthyroid subjects on the basis of hippuric acid excretion? (MUSSER *et al.*, 1955; HAM *et al.*, 1951; MANDELBROTE and WITTKOWER, 1955).

(4) With what accuracy do scores on the Taylor Manifest Anxiety Scale, measurements of blood pressure and pulse rate, and other quantitative variables predict an individual's position upon the gradient of clinical level of free anxiety? (TAYLOR, 1953; DAVIDS, 1955; FUNKENSTEIN *et al.*, 1954).

(5) Do the physical variables of body size, age, and urine volume significantly influence hippuric acid excretion in a group of subjects who vary widely in these as well as in level of free anxiety? (SCURRY and FIELD, 1943; STERN *et al.*, 1945; MACHELLA *et al.*, 1942; PERSKY *et al.*, 1950).

(6) Is a variable standard of normal hippuric acid excretion, based upon physical variables, necessary or desirable when the test is applied as an index of human free anxiety? (SCURRY and FIELD, 1943; PINCUS, 1950).

Subjects. The thirty-one individuals studied were referred by persons not directly participating in the study who attempted to include in the group a wide range of levels of free anxiety, of body sizes, and of ages. Although most subjects were seeking psychiatric therapy, the general nature of the study was known to all participants. No psychotic individuals were included. Otherwise, the group represented a heterogeneous sample ranging from five nominally healthy student volunteers through individuals with mild, moderate, and severe emotional problems, primarily on neurotic bases. The group included seven proved hyperthyroid patients who were otherwise physically well and ambulatory and were studied on an equal basis with the other subjects. All participants were free from evidence of liver disease, urinary tract disease, or nutritional inadequacy.

Procedure. All experiments were carried out in the late afternoon at least 2 hr following a light

lunch. The sequence for most subjects consisted of three parts: (1) Obtaining of personal data, cardiovascular and other measurements, and administration of the Taylor Scale. (2) Clinical interview and rating. (3) Administration of the hippuric acid test.

Each part of the experiment was carried out consistently by a different person who did not know the results of the other two parts and could not be influenced thereby. A case number was designated to identify each subject in the study.

Personal data. Extraneous factors which might influence the other values were noted and screened prior to proceeding with each interview. Factual data obtained included age, race, sex, medical history, nutritional status, medications, day of menstrual cycle, and previous experience with venipuncture and injections. Standardized measurements included height without shoes, weight without clothing, and oral temperature. Body surface area was computed from height and weight by the nomogram of DUBOIS (1927).

Immediately before and after each interview, the basal blood pressure and pulse rate were determined (FUNKENSTEIN *et al.*, 1954). TAYLOR's Manifest Anxiety Scale was administered and independently scored following the interview (1953). A routine urinalysis was done on a specimen voided following the interview.

Clinical rating. Irrespective of diagnosis or status, each subject was rated for current level of free anxiety on the basis of clinical interview. Two psychiatrists, Rater A and Rater B, rated each subject using a one-way screen and sound system to permit independent judgments based on the same interview situation, choosing at random the one who would interview the subject. These separate ratings were not compared until later. It was decided to accept the ratings of the senior observer, Rater A, as the basis of comparison for the other measurements. In two cases, technical difficulties necessitated single ratings.

A semi-structured interview protocol was designed specifically for the study and was used in each case. This served merely to focus attention systematically to a wide area of psychologic function. Items covered a cross-section of current adjustment in specific social, intrapsychic, and psychophysiologic areas. Also included for consideration were items pertaining to the personality background, neurotic traits, reality stresses, adequacy of defences, relevant medical history, the patterning of previous free anxiety, and the objective, observable, anxiety-derivative behaviour in the interview situation.

Immediately following the interview, the observers made forced quantitative ratings of the subject along a scale of level of free anxiety. The scale ranged from 0 to 6 points, where 0 represented "no free anxiety" and where 6 represented the "maximum possible rating." Both whole number and fractional (+ or −) ratings were permitted, the latter intermediate ratings being counted as thirds of a point.

In addition to the overall rating of current free anxiety, the concomitants of free anxiety were categorized and given similar subratings. The following subcategories were used: "observable," "spontaneously reported," "psychological," and "physiological" concomitants. Qualitative judgments and diagnostic formulations were also recorded for each subject.

Hippuric acid test

The intravenous one hour hippuric acid test was administered to the subjects following all other procedures and generally about 3–4 hr after a light lunch. The method used was essentially as outlined by QUICK (1939) and may be summarized as follows.

The subject is instructed to collect a urine specimen, emptying the bladder completely. Immediately thereafter, 20 cm^3 of sterile solution containing 1·77 g sodium benzoate is injected intravenously. This dose on full conjugation is equivalent to 2·20 g hippuric acid. The injection is given slowly over 5 min. At least one glass of water (180 cm^3) is then ingested. At 60 min after the completion of the injection, the subject again voids, emptying the bladder completely. In the interim he may read or sleep.

The volume of the latter urine specimen is measured at room temperature. For each 10 cm^3 of urine, 5 g of ammonium sulphate is added. The solution is filtered over suction, acidified, and stirred. The precipitate of hippuric acid is then collected over suction, washed for 30 sec with 10 cm^3 distilled water at room temperature, dried at 100°C, cooled in desiccator overnight, and weighed. The volume of the filtrate must be measured, and the quantity of hippuric acid remaining in solution, approximately 0·1 g/100 cm^3 of filtrate, is added to the weight of the precipitate. An additional 0·04 g is added for the average loss of hippuric acid occurring when the crystals are washed.

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TABLE 1.—INDIVIDUAL SUBJECT DATA

Case	Free anxiety measures: Clinical rating A	Clinical rating B	Taylor scale score	Hippuric acid (g)	Hippuric acid Index	Physical variables: Age	Race	Sex	Height (in.)	Weight (lb)	Urine volume (cm^3)
1	5−	4−	*	1·74	176	33	W	F	60½	95	195
4	4+	5	33	1·85	163	44	W	F	68	129	515
7	4	*	27	1·95	148	32	W	M	76	225	92
3	4	3	*	1·48	146	40	W	F	64	106	300
24	4−	4	39	1·45	140	37	W	F	60½	136	61
15	4−	4−	28	*	*	18	W	F	61½	99	*
22	4−	3+	36	1·26	100	48	W	M	67¾	155	235
20	3+	3+	37	1·19	111	27	W	F	65¼	140	63
17	3+	3−	20	1·58	135	47	W	F	65¼	138	275
12	3+	3−	30	*	*	59	N	M	65½	151	*
2	3−	4	*	1·55	125	37	W	M	67¼	161	93
5	2	2−	30	1·35	124	32	W	F	63	109	416
10	2−	2	32	*	*	36	W	F	62½	125	*
26	1+	1	13	1·19	108	52	W	F	61¾	133	202
29	1+	1+	5	1·62	105	23	W	M	68	161	380
8	1	2−	24	1·16	115	47	W	M	63½	125	73
16	1	2−	15	1·14	114	46	W	F	64¼	128	73
25	1	1+	19	1·24	104	32	N	F	66¾	150	83
6	1	1	12	1·76	96	19	W	M	72½	194	158
31	1−	1−	12	1·20	104	23	W	M	72½	207	61
28	+−	+−	9	1·83	114	21	W	M	70½	167	490
30	+−	+−	5	1·45	111	22	W	M	69½	150	92
27	+−	+−	3	2·09	106	22	W	M	69¾	218	320
21	+−	1−	28	1·60	104	45	W	F	64	195	360
				(Hyperthyroid patients)							
32	4+	4	22	1·32	115	52	W	F	61½	141	184
19	3−	3−	36	1·53	113	35	N	F	63½	151	275
11	3−	3−	14	*	*	25	N	F	67½	205	*
14	2	2−	15	1·28	116	58	W	M	65¾	140	136
9	*	1+	14	1·34	99	26	N	M	71	144	110
13	+−	+−	20	*	*	57	W	M	69½	151	*
18	+−	1	*	1·08	100	58	W	M	65¾	136	126

* Data not obtained.

RESULTS

Table 1 presents the principal individual subject data. For convenience of analysis, the subjects are arranged in descending order of clinical free anxiety rating. The hyperthyroid patients are grouped in a similar order at the bottom of the table. Technical difficulties prevented the accumulation of all data on each subject.

The data regarding hippuric acid is presented in two ways: (1) as grammes excreted, and (2) as an index, which expresses excretion as a percentage of the estimated normal excretion for each subject. The latter involves a concept to be expanded subsequently.

Correlational data

Figure 1 demonstrates graphically the degree of relationship found between the independent clinical ratings of free anxiety. Although not complete, the agreement between the raters is very close. The nature of the variables, continuous but relative,

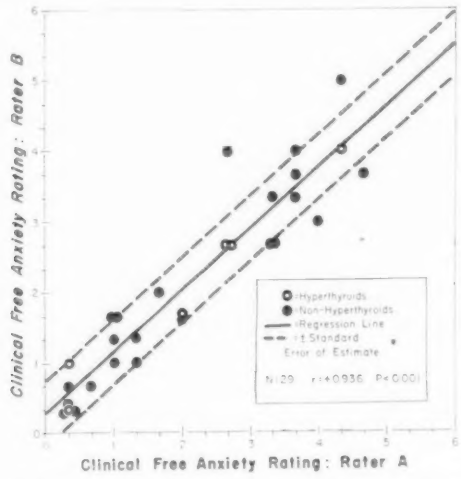

FIG. 1.—Relationship between independent clinical ratings of free anxiety.

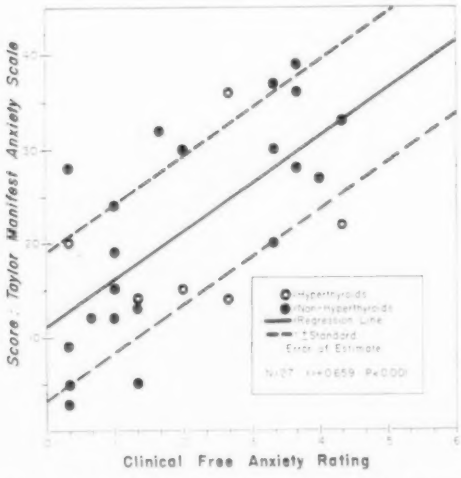

FIG. 2.—Comparison of Taylor Manifest Anxiety Scale with clinical ratings.

makes the coefficient of correlation (r) an adequate measure of the relationship (EZEKIEL, 1941). This coefficient equals +0·94 and is very highly significant.

Figure 2 illustrates the comparison of the Taylor Manifest Anxiety Scale scores with the clinical free anxiety ratings. The coefficient of correlation equals +0·66 and is highly significant. The best measure of the accuracy of the scale, however, is the

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standard error of estimate (EZEKIEL, 1941). This measure reflects the rather wide spread of scores found on the scale.

No significant relationships were found between the clinical free anxiety ratings and such quantitative variables as basal blood pressure and pulse rate, including net differences before and after the interview. These data, consequently, are not presented.

Figure 3 illustrates the relationship found between hippuric acid excretion and the clinical free anxiety ratings, both excluding and including the hyperthyroid patients. Hippuric acid was found to be so slightly related to the clinical rating that the overall relationship fell far short of statistical significance.

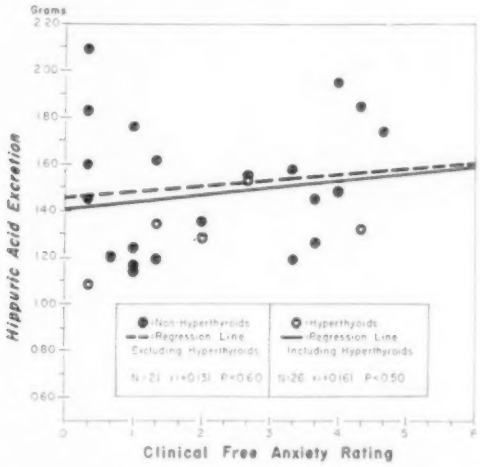

FIG. 3.—The hippuric acid test and clinical ratings of free anxiety.

This failure to confirm previous findings by other authors may be caused by various factors, such as differences in population tested (homogeneity of previous studies versus heterogeneity of this study), different procedures in evaluating anxiety, and others. The high degree of differences in physical variables seems to obscure the relationship existing between free anxiety and hippuric acid excretion.

This finding is not due to the inclusion of hyperthyroid patients in the subject group (PERSKY *et al.*, 1952; BASOWITZ *et al.*, 1955; MUSSER *et al.*, 1955; HAM *et al.*, 1951; MANDELBROTE and WITTKOWER, 1955). The correlation between the clinical rating and hippuric acid is even more reduced when the hyperthyroid patients are excluded from consideration. The inspection of the data in Table 1 shows that the hippuric acid test values, furthermore, do not clearly differentiate this small group of hyperthyroid patients from the non-hyperthyroid subjects.

Figure 4 gives a composite graphic illustration of the association between weight, age, and urine volume and hippuric acid excretion. The relationship between each of these physical variables and hippuric acid excretion is statistically significant below the 5 per cent level of confidence. Body surface area is related to hippuric acid excretion with a coefficient of correlation of +0·45 which is significant at a slightly higher confidence level than is body weight. Because of the slight difference, only the simpler measure of body weight, is illustrated. These findings are consistent with the evidence

from a number of previous studies (SCURRY and FIELD, 1943; STERN *et al.*, 1945; MACHELLA *et al.*, 1942; PERSKY *et al.*, 1950).

It is possible to demonstrate by the technique of partial correlation that a significant relationship, actually present between hippuric acid excretion and clinical free anxiety rating, has been obscured by the composite influence of the physical variables EZEKIEL, 1941). Ezekiel writes: "It is evident that a mere surface examination of a set of data cannot reveal which independent factors are important and which are unimportant. A variable which shows no correlation with the dependent variable may

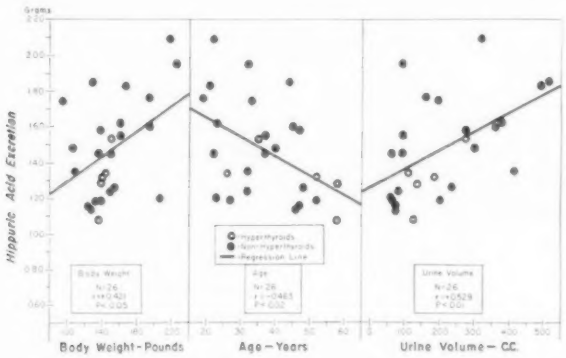

FIG. 4.—Three factors normally influencing the hippuric acid test.

yet show significant correlation after the relation to the other variables has been allowed for."

The intercorrelations between hippuric acid excretion, clinical free anxiety rating, body weight, age, and urine volume are presented in the upper part of Table 2. The partial correlations between each of these independent variables, holding constant the net effects of the other independent variables, and hippuric acid are on the last line of the table. The magnitude of these correlations approach the ones found in previous studies and confirms the hypothesis that physical variables obscure the relationship.

The coefficients of partial correlation show that the influence of weight and urine volume upon hippuric acid excretion are both highly significant in this sample (above 0·05 level). The residual influence of age is significant only at the 0·10 level of confidence. The coefficient of partial correlation in the sample, between hippuric acid excretion and clinical free anxiety rating, is +0·62, which is significant at the 0·005 level of confidence.

Derivation of the hippuric acid index

Although the analysis presented above permits us to determine the net strength of each relationship in the sample, they do not explain in what way the independent variables interact to determine hippuric acid excretion. Accordingly, further analysis of the data was undertaken (EZEKIEL, 1941). Exhaustive graphic analysis of the relationship between hippuric acid excretion and the independent variables indicated the following:

(1) A complex joint function of a multiplicative type appears likely between the independent variables and hippuric acid excretion.

(2) Use of the formula of SCURRY and FIELD, to predict individual normal values

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of hippuric acid excretion based on body weight, appears to provide an adequate base line to hold constant the influence of body size (SCURRY and FIELD, 1943).

(3) The net effect of urine volume appears curvilinear, low volumes tending to decrease hippuric acid excretion sharply, but high volumes tending to have relatively little effect. The principle used to correct urea clearance determinations for low urine volumes closely fits the data of the present study (MOLLER *et al.*, 1928; MCINTOSH *et al.*, 1928; SMITH *et al.*, 1945; KOHLSTAEDT and HELMER, 1936). It is known empirically that the clearance varies in proportion to the square root of the urine volume below a

TABLE 2.—RELATIONSHIPS AMONG MAJOR VARIABLES

	Anxiety rating	Weight	Age	Urine volume
Simple correlation coefficients				
Hippuric acid	+0·16	+0·42†	−0·46†	+0·52‡
Anxiety rating		−0·36*	+0·25	+0·05
Weight			−0·42†	−0·05
Age				−0·07
Partial correlation coefficients				
Hippuric acid	+0·62‡	+0·82§	−0·39*	+0·74§
Confidence level ($N = 26$)				

* 0·10
† 0·05
‡ 0·01
§ 0·001

variable point termed the "augmentation limit," each measurement being first adjusted to a standard surface area. Above this point, clearance is unaffected by further increases in urine volume. By analogy, a minimum urine volume may be postulated for each individual based upon his surface area, below which a similar square root relationship appears to hold. The use of this principle as a correction factor would seem to provide an adequate means of holding constant the net influence of urine volume.

(4) The residual effects of age amount to less than 1 per cent decrease in hippuric acid excretion per year of age when standardized to age 35. A simple age correction factor appears to provide an approximate means of holding constant the net influence of age.

(5) The net effect of free anxiety upon hippuric acid excretion also appears to be curvilinear, as determined by the clinical ratings.

Increases in level of free anxiety at low levels exert minimal influence upon the test or actually decrease the values. Further increases in level of free anxiety, however, are associated with increasingly sharp rises in hippuric acid excretion.

These correlational functions can be incorporated into a mathematical construct which expresses the estimated excretion of hippuric acid for each subject that can be expected on the basis of the physical factors. The derived parameter provides an index of the "normal" standard against which to evaluate the residual influence of free anxiety. That is, estimating formulas may be used to hold constant the influence of the physical variables (SCURRY and FIELD, 1943; PINCUS, 1950).

Application. The "hippuric acid index" is the term used to designate hippuric acid excretion expressed as a per cent of the estimated normal excretion:

$$\text{hippuric acid index} = \frac{\text{hippuric acid excretion (g)} \times 100}{\text{(corrected) normal hippuric acid (g)}}$$

Normal hippuric acid excretion, with adequate urine volume, is estimated as the product of two factors, a weight factor and an age factor. From the prediction equation of SCURRY and FIELD (1943), for hippuric acid in grammes:

$$\text{weight factor} = 0{\cdot}34 + (0{\cdot}00668 \times \text{weight (lb)})$$

$$\text{age factor} = 150/\text{age (yr)} + 115$$

The correction factor for inadequate urine volume becomes the third factor multiplied to estimate normal hippuric acid excretion, but only in certain cases is this correction required. It is required only when body size is large or when urine volume is small, each measure in effect adjusted to a standard surface area. The minimum urine volume for each person is estimated as

$$\text{minimum urine volume (cm}^3\text{)} = 34{\cdot}7 \times (\text{surface area (m}^2\text{)})$$

Only when the urine volume, during the 65 min test interval, is less than the minimum urine volume is the correction factor applied, estimated as:

$$\text{urine volume factor} = \left(\frac{\text{urine volume (cm}^3\text{)}}{\text{minimum urine volume (cm}^3\text{)}}\right)$$

The derived variable standard of normal, which estimates each person's normal hippuric acid excretion with the 1 hour intravenous test, is:

$$\text{(corrected) normal hippuric acid (g)} = \begin{pmatrix}\text{weight}\\ \text{factor}\end{pmatrix} \times \begin{pmatrix}\text{age}\\ \text{factor}\end{pmatrix} \times \begin{pmatrix}\text{urine volume factor,}\\ \text{when inadequate}\end{pmatrix}$$

Nomograms. To simplify the calculation of the hippuric acid index, two nomograms have been prepared, based upon the above estimating formulas.

Figure 5 gives a nomogram which incorporates the weight factor and the age factor and permits rapid estimation of normal hippuric acid excretion.

Figure 6 gives a nomogram which incorporates minimum urine volume and the correction factor for use when urine volume is inadequate. Ordinarily, if the 65 min urine volume exceeds 200 cm³ or if body size is small relative to urine volume, this factor may be disregarded. Otherwise, body surface area is computed from height and weight (DU BOIS, 1927). From surface area, the minimum urine volume may be read off directly from the scale along the right. With inadequate urine volume, a straight line connecting the proper points for urine volume and surface area crosses the middle line at the point for the estimated correction factor.

Table 3 presents an itemized account of the steps used in the calculation of the hippuric acid index for the subjects on whom data are complete. Through the estimating formulae or by use of the nomograms prepared, the individual subject data of Table 1 may be followed through the calculation of the index.

Increase in accuracy. Figure 7 demonstrates graphically the relationship between the hippuric acid index and the clinical ratings of free anxiety, both excluding and

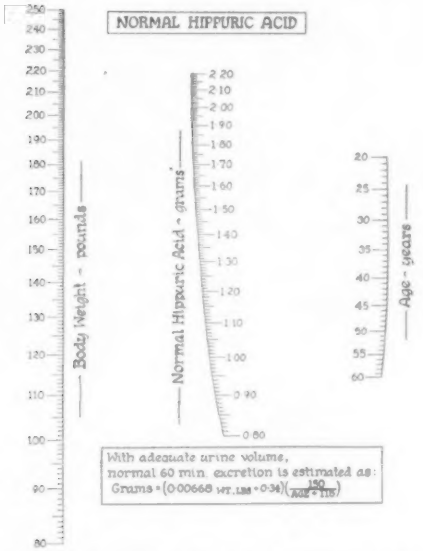
NORMAL HIPPURIC ACID
Body Weight - pounds
Normal Hippuric Acid - grams
Age - years
With adequate urine volume, normal 60 min. excretion is estimated as:
Grams = (0·00668 WT.LBS + 0·34)(150 / AGE + 119)

FIG. 5.—Nomogram to calculate normal hippuric acid excretion in the 1 hr intravenous test.

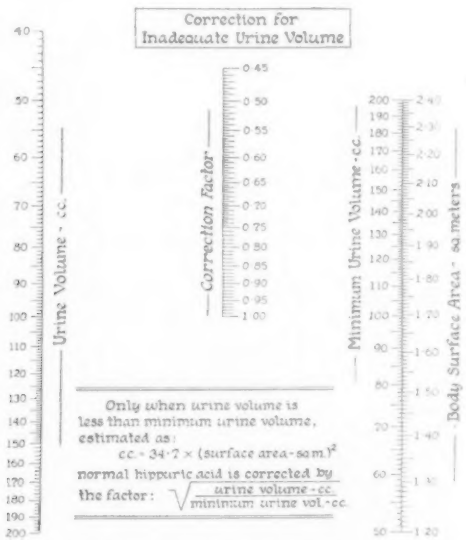
Correction for Inadequate Urine Volume
Urine Volume - cc.
Correction Factor
Minimum Urine Volume - cc.
Body Surface Area - sq meters
Only when urine volume is less than minimum urine volume, estimated as:
cc. = 34·7 × (surface area - sq m.)²
normal hippuric acid is corrected by the factor: √(urine volume - cc. / minimum urine vol. - cc.)

FIG. 6.—Nomogram to calculate minimum urine volume and correction factor for inadequate urine volume.

including the hyperthyroid patients. The agreement between the index and the clinical ratings is very close in both instances but is greater with the hyperthyroid patients excluded from consideration. The curvilinear relationship evident between level of free anxiety and the hippuric acid index may be expressed approximately by a parabolic regression curve (EZEKIEL, 1941). The two curves illustrated have been mathematically determined by the usual methods. The coefficients of correlation

based upon these are +0·86 and +0·79, excluding and including the hyperthyroid patients, respectively. Despite the number of constants involved, for which the usual adjustments have been made, these coefficients are very highly significant at the 0·001 level of confidence (EZEKIEL, 1941).

The subjects having hippuric acid index values of 120 per cent or above are

TABLE 3.—CALCULATION OF HIPPURIC ACID INDEX*

Case	Weight factor	Age factor	Urine volume factor	Surface area (m^2)	Minimum urine vol. (cm^3)	(Corrected) normal hippuric acid (g)	Hippuric acid (g)	Index % of normal
1	0·975	1·01		1·35	63	0·988	1·74	176
4	1·202	0·94		1·69	99	1·134	1·85	163
7	1·843	1·02	0·702	2·32	187	1·320	1·95	148
3	1·048	0·97		1·49	77	1·014	1·48	146
24	1·248	0·99	0·839	1·58	87	1·034	1·45	140
22	1·375	0·92		1·82	115	1·265	1·26	100
20	1·275	1·06	0·793	1·70	100	1·067	1·19	111
17	1·262	0·93		1·68	98	1·168	1·58	135
2	1·415	0·99	0·890	1·84	118	1·242	1·55	125
5	1·068	1·02		1·49	77	1·090	1·35	124
26	1·228	0·09		1·59	88	1·103	1·19	108
29	1·415	1·09		1·85	119	1·538	1·62	105
8	1·175	0·93	0·924	1·57	86	1·005	1·16	115
16	1·195	0·93	0·901	1·61	90	1·003	1·14	114
25	1·342	1·02	0·874	1·77	109	1·197	1·24	104
6	1·636	1·12		2·12	156	1·831	1·76	96
31	1·723	1·09	0·614	2·16	162	1·150	1·20	104
28	1·456	1·10		1·93	129	1·606	1·83	114
30	1·342	1·09	0·890	1·83	116	1·307	1·45	111
27	1·796	1·09		2·15	160	1·966	2·09	106
21	1·643	0·94		1·93	129	1·540	1·60	104
					(Hyperthyroid patients)			
32	1·282	0·90		1·63	92	1·151	1·32	115
19	1·349	1·00		1·72	103	1·349	1·53	113
14	1·275	0·87		1·71	101	1·106	1·28	116
9	1·302	1·06	0·978	115	115	1·355	1·34	99
18	1·248	0·87		1·68	98	1·082	1·08	100

* Decimals rounded for presentation.

invariably those with a moderately severe or greater level of free anxiety, as determined by the clinical ratings. Below 120 per cent, the index appears to lose accuracy, blending into what appears to be a range of normal or unexplained variation. A comparison of this range with the corresponding range of values illustrated in Fig. 3 demonstrates a considerable reduction in unexplained variation.

The small group of hyperthyroid patients is distributed entirely within the range of index values below 120 per cent, regardless of clinical free anxiety rating. Similarly, two non-hyperthyroid subjects, case 22 and case 20, clearly represent false negatives on the hippuric acid index. The possible explanation for the latter two index values may

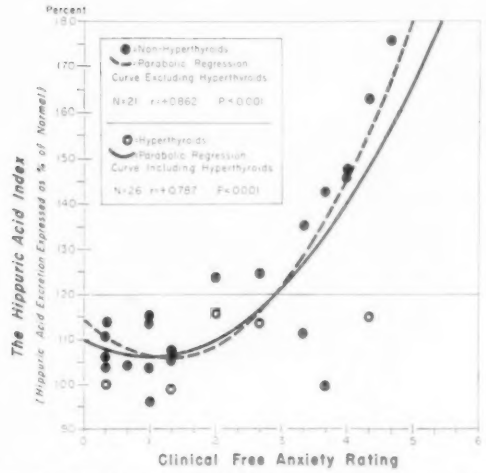

FIG. 7.—The hippuric acid index and clinical ratings of free anxiety.

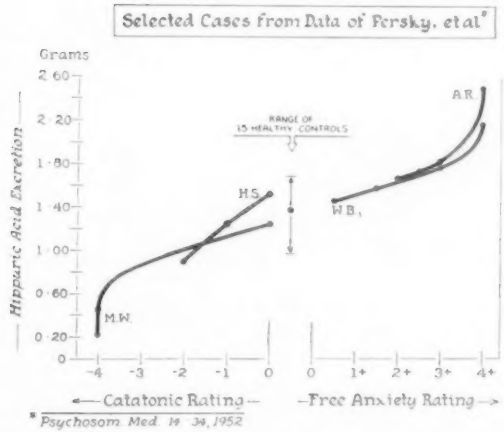

FIG. 8.—Changes in hippuric acid excretion with fluctuations in catatonia and free anxiety.

lie in incomplete voiding of urine when instructed to do so, or in metabolic factors as yet unknown.

COMMENT

Support for the validity of these conclusions is given by a phenomenon derived on consideration of a previous independent study. The individual subject data of selected cases demonstrate a remarkable parallel to the derived data of the hippuric acid index (PERSKY *et al.*, 1952).

Fig. 8 demonstrates graphically changes in hippuric acid excretion with fluctuations in catatonia and free anxiety, in selected cases from the data of PERSKY, *et al.* Changes over time, in the cases of A.R. and W.B.1, in both clinical free anxiety scores and in hippuric acid excretion, are given. These changes show the same general pattern of curvilinear response within the individual as that evident in the hippuric acid index

comparing different individuals. Also illustrated are apparently analogous, but inverse, relationships of curvilinear response between clinical catatonic scores of cases H.S. and M.W. and hippuric acid excretion.

The results and application of our preliminary study suggest that the hippuric acid test with certain refinements may be useful as an objective and quantitative measure of level of free anxiety in psychiatric patients. A variable standard of normal would appear necessary and desirable to maximize the accuracy of the index. The tentative estimating formulae given appear to provide an index of free anxiety, of which the accuracy in the present sample approaches the degree of agreement found with independent ratings by a second trained psychiatric observer.

The limitations of the present study, however, require appropriate cautions. A larger and independent series of cases would be desirable to substantiate the application of the hippuric acid index. The estimating formulae given are approximations only, doubtless capable of further refinement. Extraneous factors which may influence test values, including selection of procedure and technique of chemical determination, must also be considered. Until further studies are carried out, generalizations beyond the limits of the present data are not warranted.

SUMMARY

The hippuric acid test and other measures were studied in a heterogeneous group of selected subjects. These persons were clinically rated for level of free anxiety with close agreement between independent raters. Previous reports of hyperexcretion of hippuric acid corresponding to increasing levels of free anxiety were confirmed only when allowance was made for the effects of physical variables. From prior studies and the current data, formulae were evolved to estimate each person's normal hippuric acid excretion. The hippuric acid index designates hippuric acid excretion expressed as a percentage of this variable standard of normal. The index shows close correlation with the clinical ratings at a very high level of significance. These findings are taken to indicate that the hippuric acid test, with certain refinements, may be applied as an objective and quantitative measure of free anxiety in psychiatric patients.

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THE PROBLEM OF SPECIFICITY IN PSYCHOSOMATIC ILLNESS

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IT is apparent from the psychiatric literature that agreement has not yet been reached on the question of the specificity of psychosomatic illness. One school of thought, with a considerable following, postulates specificity in one form or another. DUNBAR (1943) and ALEXANDER (1943), for example, emphasize personality specificity, maintaining that different personality patterns underlie such psychosomatic conditions as peptic ulcer, asthma and functional heart disease. PARNELL (1955), through his studies of the somatotypes of students with psychosomatic disease expresses the view that the specific form of the illness is influenced by the constitutional endowment of the individual. The opposing school of thought is represented by STEVENSON and MATTHEWS (1953) who feel that the evidence is in favour of there being a fairly universal neurotic personality pattern underlying all psychosomatic conditions. KUBIE (1954) is in essential agreement with this view, stating that neither on logical grounds nor from his clinical experience can he accept the idea of symptomatic specificity.

WOLFF (1947) in his excellent discussion of the whole problem introduces a third concept and summarizes these three main viewpoints as follows:

(1) That there are no exclusive relationships between personality, constitutional and environmental factors on the one hand and the site of disturbance of somatic function on the other;

(2) That psychosomatic disturbance localizes in an organ or system which has some symbolic significance in the personality structure or which has been weakened by constitutional or environmental factors. This viewpoint is often referred to as the target-organ or organ-inferiority concept;

(3) That the specificity of psychosomatic disease is not in terms of anatomical location but rather in terms of the nature of the functional response. For example, the gastro-intestinal system may in one person be the site of an 'offensive' psychosomatic response characterized by hyper-acidity and ulcer formation, while in another person it may be the site of a defensive response characterized by vomiting and diarrhoea.

Of the three viewpoints, the latter is the one favoured by WOLFF. Each of these views gives rise to certain predictions.

The first implies that the presence of a psychosomatic disorder of one system will not reduce, or may even increase, the likelihood of the individual developing psychosomatic disturbances of another system.

The second view implies that an individual will tend to have his psychosomatic disturbances restricted to one organ or system.

The third view leads to essentially the same prediction as the first, but with one modification, namely that in persons with multiple psychosomatic afflictions the functional response would be similar in the various affected systems.

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The study of the long-term illness experience of individuals would permit one to see which of these predictions accords best with the observed facts. The present paper is the report of such a study, based upon records of medically attended illness over a 5 year period for 187 adults.

SOURCE OF DATA AND METHODS

The data came from a 5 per cent systematic sample of the medical records of subscribers to a pre-paid plan for medical care in an industrial Canadian city (Windsor Medical Services, Windsor, Ontario). Since the plan is extremely comprehensive, its records cover virtually all the physician-attended illnesses of its subscribers.

TABLE 1.—AGE* AND SEX SPECIFIC PROPORTIONS OF PERSONS WITH SELECTED PSYCHOSOMATIC AND PSYCHONEUROTIC DISORDERS OVER A 5-YEAR PERIOD

		Males		Females	
		20–39	40–59	20–39	40–59
	No.	52	43	52	40
With one or more:					
Allergic disorders	No.	6	5	12	7
	%	11·54	11·63	23·07	17·50
Cardiovascular disorders	No.	7	5	6	8
	%	13·46	11·63	11·54	20·00
Gastro-intestinal disorders	No.	11	9	14	11
	%	21·15	20·93	26·92	27·50
Musculo-skeletal disorders	No.	9	9	7	10
	%	17·31	20·93	13·46	25·00
Non-somatic neurotic disorders	No.	8	5	14	9
	%	15·38	11·63	26·92	22·50

* Refers to age in 1948

The 187 individuals who form the basis of the present report were all those in the sample who enrolled in the plan in 1946, who were aged 20–59 years in 1948 and had records for 5 complete years between 1948 and 1953.*

For each individual, illnesses were transcribed from the original records, coded to the sixth revision of the International Statistical Classification (1948) and then numbered as illness episodes according to a set of arbitrary rules. The principle underlying these rules was to count as one episode all symptoms, complications and therapeutic services believed to relate essentially to one illness. By this means, inflated illness counts were avoided and the advantage of the longitudinal record in minimizing errors connected with vague or changing diagnoses was exploited.

* Records with sufficient diagnostic information were not kept by Windsor Medical Services until 1948.

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For the present analysis five categories of psychosomatic or psychoneurotic illness were considered:

(1) Allergic disorders.
(2) Psychosomatic disorders of the cardiovascular system.
(3) Psychosomatic disorders of the gastro-intestinal system.
(4) Psychosomatic disorders of the musculo-skeletal system.
(5) Behavioural and character disorders and non-somatic psychoneuroses.

The specific diagnoses included in each of these categories are shown in the Appendix.

The first step in the analysis was to determine the proportion of persons who were attended for one or more conditions in each of the five categories. These proportions, calculated on an age and sex-specific basis are shown in Table 1. At first glance they appear surprisingly high, but it must be remembered that they refer to a 5 year period of risk rather than the time period of 1 year customarily used for the expression of morbidity statistics.

The next step was to take the ten possible pairs of the five categories of psychosomatic and psychoneurotic illness and to calculate for each pair, the expected number of persons with illness in both categories, in neither category and in one category only. The calculation of these expected numbers involved the use of the age and sex specific proportions shown in Table 1. An example will make the method clear. For the pair—allergies and cardiovascular disorders—the expected numbers in the fifty-two males aged 20–39 were obtained as follows:

Expected number with allergy only:	
0·1154 × 0·8654 × 52 =	5·19
(proportion of males 20–39 with allergy, times proportion of males 20–39 without cardiovascular disorders, times total number of males 20–39)	
Expected number with cardiovascular disorders only:	
0·1346 × 0·8846 × 52 =	6·19
Expected number with both allergy and cardiovascular disorders:	
0·1154 × 0·1346 × 52 =	0·81
Expected number with neither allergy nor cardiovascular disorders:	
0·8846 × 0·8654 × 52 =	39·81

The expected numbers for males aged 40–59 and for females aged 20–39 and 40–59 were calculated similarly and then a total obtained by summation of the values for the four age–sex groups. This procedure was carried out for each of the ten pairs of categories.

It is desirable to make clear at this point, exactly what is meant by the expected number. It is the number which one would expect if the five categories of diseases occurred absolutely independently. Any significant departure from expectation can be taken as evidence that independence does not hold. If the observed number of persons with two particular psychosomatic disorders were *less than expected*, one would take this to mean that the occurrence of the one disorder tended to preclude the occurrence of the other. If, on the other hand, the observed number of persons with two particular disorders were *greater than expected*, one would conclude that the occurrence of the one disorder increased the likelihood that the other would occur.

TABLE 2.—OBSERVED AND EXPECTED NUMBERS OF PERSONS WITH VARIOUS COMBINATIONS OF TEN PAIRS OF PSYCHOSOMATIC AND PSYCHONEUROTIC DISORDERS (MALES + FEMALES, AGED 20–59)

Pair	Combination	Observed No.	Expected No.	X^2	d.f.*	Probability of diff. being due to chance
Allergy	All., no C.V.	25	25·82			>0·50
and	C.V., no All.	21	21·83	0·2278	1	not
cardiovascular	All. + C.V.	5	4·17			significant
	neither	136	135·18			
Allergy	All., no G.I.	17	22·54			<0·02
and	G.I., no All.	32	37·54	6·5503	1	significant
gastro-intestinal	All. + G.I.	13	7·46			
	neither	125	119·46			
Allergy	All., no M.S.	22	24·55			>0·10
and	M.S., no All.	27	29·55	1·7408	1	not
musculoskeletal	All. + M.S.	8	5·44			significant
	neither	130	127·45			
Allergy	All., no neur.	20	23·69			<0·10 > 0·05
and	neur., no All.	26	29·69	3·2982	1	borderline
non-somatic neurosis	All. + neur.	10	6·31			significance
	neither	131	127·31			
Cardiovascular	C.V., no G.I.	11	19·66			<0·001
and	G.I., no C.V.	30	38·66	18·1965	1	highly
gastro-intestinal	C.V. + G.I.	15	6·34			significant
	neither	131	122·34			
Cardiovascular	C.V., no M.S.	17	20·94			<0·05
and	M.S., no C.V.	26	29·94	4·4462	1	significant
musculoskeletal	C.V. + M.S.	9	5·06			
	neither	135	131·06			
Cardiovascular	C.V., no neur.	13	20·92			<0·001
and	neur., no C.V.	23	30·92	17·8570	1	highly
non-somatic neurosis	C.V. + neur.	13	5·08			significant
	neither	138	130·08			
Gastro-intestinal	G.I., no M.S.	33	36·58			>0·10
and	M.S., no G.I.	23	26·59	2·4792	1	not
musculoskeletal	G.I. + M.S.	12	8·41			significant
	neither	119	115·41			
Gastro-intestinal	G.I., no neur.	26	36·02			<0·001
and	neur., no G.I.	17	27·02	18·5568	1	highly
non-somatic neurosis	G.I. + neur.	19	8·98			significant
	neither	125	114·98			
musculoskeletal	M.S., no neur.	24	28·45			
and	neur., no M.S.	25	29·44			<0·05
non-somatic neurosis	M.S. + neur.	11	6.55	4·5511	1	significant
	neither	127	122·55			

* 1 degree of freedom, because in addition to removal of 1 d.f. for linear constraint, 1 d.f. is removed for each of the two proportions used to calculate the expected values.

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RESULTS

Table 2 presents the observed and expected numbers for each of the ten pairs of categories and gives the X^2 values which permit assessment of the statistical significance of differences between observed and expected.

In six of the ten pairs there is a significant excess of individuals with *both* disorders; allergy and gastro-intestinal disorders, cardiovascular and gastro-intestinal disorders, cardiovascular and musculo-skeletal disorders, cardiovascular disorders and non-somatic neuroses, gastro-intestinal disorders and non-somatic neuroses and musculo-skeletal disorders and non-somatic neuroses.

For one pair, allergy and non-somatic neuroses, there is an excess of persons with both disorders which attains a borderline level of statistical significance.

In the remaining three pairs, the observed and expected distributions do not differ significantly, but for none of these pairs is there a deficit of persons with both disorders.

DISCUSSION

These results lend no support to the belief that psychosomatic disorders tend to be restricted to one bodily system within a given individual. The very reverse, in fact, appears to be the case. When the five categories of illness are taken two at a time, there are invariably more people with disorders in both categories than would be expected if the occurrence of each were independent and for six out of the ten pairs this excess is statistically significant.

In other words, there is no evidence of organ or system specificity, but rather of a tendency for psychosomatic illness to be diffuse in its manifestations within the individual. It should be pointed out again that the 187 individuals studied were followed for a period of 5 years, a somewhat longer period than is usual in a systematic investigation of illness. During any particular interval of time within the 5 year period it is highly possible that only one psychosomatic or neurotic disorder was present. Thus an impression of psychosomatic specificity might easily be gained by a physician who did not have an opportunity to observe his patient over a sufficiently lengthy period. (One could, of course, take the view that a degree of specificity might exist over short periods, but that with a change in either environmental or psychological factors there could be a change in the individual's specific predisposition.)

At the beginning of this paper, mention was made of WOLFF's point of view that specificity might exist in terms of functional response rather than in terms of organs or systems. This possibility has not, of course, been eliminated by the results of the present investigation. Although multiple psychosomatic disorders have been observed significantly more frequently than the hypothesis of coincidence would predict, no evidence has been offered for or against the occurrence within the individual of a specific functional response in various systems. This possibility, which is of great interest, has not been examined because of the difficulty of classifying functional responses. With our present knowledge, an attempt to set up such a classification would be premature. However, it is to be hoped that further experimental work in the field of psychosomatic medicine will suggest a reasonable basis for such a classification. When this time comes, an analysis similar to the present one, but based upon a functional rather than a system classification, should be carried out.

The present study has not attempted to go beyond the investigation of the frequency of pairs of psychosomatic disorders. Taking three or more disorders at a time

is theoretically quite feasible but was not practical with the size of the present sample, since both the expected and observed numbers would become extremely small.

The significant associations which have been observed between particular pairs of disorders are of considerable interest and it should be mentioned that two of these associations have been observed by another worker. GOSLING (1958) has recently reported upon his finding of an excessive incidence of peptic ulcer in persons with neuroses (excluding cardiovascular and gastro-intestinal neuroses) and in asthmatic patients.

SUMMARY

A statistical study has been made of the frequency of multiple psychosomatic and psychoneurotic disorders in 187 adults observed over a 5 year period. The results do not conform to the hypothesis of single organ or single system specificity. They suggest instead that multiple psychosomatic disorders occur significantly more often than would be expected on the basis of pure coincidence.

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APPENDIX

List of illnesses included in each of the five categories of psychosomatic and psychoneurotic disorders

ISC No.*	Diagnosis	No. of cases
	Allergic disorders	
240	Hay fever	8
243	Urticaria	8
241	Asthma	7
242	Angioneurotic oedema	5
244–245	Other allergies	4
	Cardiovascular disorders	
444	Essential benign hypertension without heart disease	11
315	Cardiac neurosis	5
782	Cardiovascular symptoms	5
467·0	Hypotension	4
433·1	Other disorders of heart rhythm	1
	Gastro-intestinal disorders	
573	Functional disorders of the intestines	13
541	Duodenal ulcer	10
784	Upper gastro-intestinal tract symptoms	7
316	Gastric neurosis	5
544	Disorders of function of the stomach	4
543	Gastritis and duodenitis	3
585	Cholecystitis without calculi	2
552	Chronic appendicitis	2
	Musculo-skeletal disorders	
726–727	Muscular rheumatism	22
725	Arthritis unspecified	13
	Non-somatic neuroses	
790	Nervousness and debility	17
310	Anxiety neurosis	13
318	Neurasthenia	7
321·0	Emotional upset	1
302	Involutional melancholia	1

* Three-digit number in the International Statistical Classification of Diseases, Injuries and Causes of Death, sixth revision, 1948.

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THE ROLE OF EMOTIONAL AND ALLERGIC FACTORS IN HAY FEVER

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HAY fever may be regarded as a prototype for all allergic disorders. It is, by definition, an allergic reaction to pollen and has positive cutaneous reactions to pollens as a characteristic feature. FREEMAN (1950) considered that hay fever provided a key to the understanding of allergic disorders in general. It is, therefore, particularly important to know what role, if any, psychological factors may play in the disorder. The role of suggestion has been given considerable prominence in the literature by repeated quotations of instances of hay fever symptoms being precipitated by artificial flowers, pictures of hayfields, etc. Suggestion, however, is only one of many possible psychological factors which may be relevant in hay fever.

There is considerable controversy regarding the nature and incidence of psychological factors in hay fever. Some authors consider personality to be a predisposing cause and describe more or less specific personality types associated with hay fever (WITTKOWER, 1938; STOKES and BEERMAN, 1940). Other authors claim that there are specific psychodynamic processes in the pathogenesis of hay fever (DUNBAR, 1938; WILSON, 1941). Furthermore, little agreement exists regarding the role played by emotional changes and stressful life situations in precipitating or predisposing to attacks of hay fever.

The present study was carried out with the object of obtaining information in answer to the following questions.

(1) Whether hay fever patients exhibited specific personality types or patterns.

(2) Whether hay fever patients as a group differed from a comparable control group in the incidence of neurosis or neurotic symptoms.

(3) Whether emotional changes played any role in hay fever either at its onset or during its course?

MATERIAL

The groups studied included a random sample of fifty hay fever subjects attending the Asthma and Allergy Clinic of St. David's Hospital, Cardiff and a control group of fifty patients who had herniotomy or appendicectomy operations, matched for age and sex distribution. Both hay fever patients and controls were investigated and assessed by the author using identical methods, standards and criteria. A record sheet of some 200 items relating to data on social and family history, personal history, personality and other clinical and laboratory findings was completed for each hay fever patient and control subject. The information was eventually transferred to Hollerith punched cards and analysis carried out by the Hollerith Counter Sorter.

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DIAGNOSTIC CRITERIA

The symptoms of hay fever consist of sneezing, running and blocking of the nose, irritation of the eyes, nose, ears and throat. Inflammatory changes are found in the eyeball, nose and throat and sometimes bronchi (FREEMAN, 1950). The diagnosis of hay fever is based on:

(1) Accurate history.

(2) The occurrence of symptoms during the time the atmosphere contains relatively large quantities of pollen.

(3) Examination of the nasal mucosa. The appearance is typically greyish white with a water-logged, boggy appearance.

(4) Blood counts.

(5) Examination of nasal smears.

(6) Skin tests with pollens.

These criteria and methods were applied to all patients in the group.

CLINICAL DATA

Age and sex distribution

The group consisted of 36 per cent males and 64 per cent females. The age at the time of examination was distributed as follows: 0–15 years 2 per cent; 15–25 years 34 per cent; 26–35 years 32 per cent; 36–45 years 12 per cent; 46–55 years 12 per cent; 55 years+ 8 per cent.

Age at onset. The age of initial manifestations of hay fever symptoms ranges from infancy to old age. In this group the age at onset was distributed as follows: 0–5 years 6 per cent; 6–15 years 38 per cent; 16–25 years 30 per cent; 26–35 years 14 per cent; 36–45 years 6 per cent; 46–55 years 6 per cent.

Nearly three-quarters of the patients had an onset of hay fever before 25 years of age. The nature of the factors determining the actual age at onset are not fully known. Whether genetic or constitutional factors or the degree and duration of exposure to pollen are primarily responsible or by other influences is not known and further research is needed to elucidate this important problem.

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Personality

STOKES and BEERMAN (1940) in a review of the literature concluded that there was a growing impression among investigators that the allergic subject exhibited a more or less distinct personality pattern. WITTKOWER (1938) in one of the few studies in which adequate controls were used, found marked differences between a group of fifty hay fever patients and a control group consisting of twenty-seven patients with acute appendicitis. He found out that in personality, hay fever patients tended to be shut in, self absorbed, shy, aloof, very sensitive and easily discouraged. At work they were meticulous, conscientious, perfectionistic and ambitious. He found that the incidence of gross neurotic symptoms was higher in the hay fever group than in the control series.

Psychoanalytic studies on three hay fever subjects were reported by DUNBAR (1938) who found that sexualization of respiratory function and great interest in the sense of smell was a feature in these patients. Compulsive tendencies with ambivalence were also noticed, with a tendency to cyclothymic behaviour in symptom free periods. WILSON (1941) reported on psychoanalytic study of seven patients and claimed that unsatisfactory olfactory repression was closely related to hay fever. These psychoanalytic studies are based on very small groups, which are unlikely to be random samples of the disorder and it is considered extremely doubtful whether it is justifiable to apply these conclusions to hay fever in general.

In the present study hay fever patients and controls were rated by the author on a three point scale with regard to a number of personality traits, employing the same standards and criteria for each group.

From Table 1 it will be seen that a variety of personality traits were found in the

hay fever group. No specific personality pattern was found. The only differences between the hay fever and control group, which are statistically significant (i.e. with a critical ratio of two or more), are traits rated as very anxious, very ambitious and very obsessional. There is no evidence that hay fever patients were more unstable, maladjusted, or cyclothymic in temperament, as has been claimed.

TABLE 1

Personality traits	Hay fever group (%)	Control group (%)
General stability and adjustment		
stable	72	82
moderately unstable	20	15
very unstable	8	2
Meek and dependent		
somewhat	40	35
very	16	6
Sensitive		
somewhat	26	21
very	40	37
Anxious		
somewhat	36	25
very	46	27
Meticulous, obsessional		
somewhat	36	25
very	46	26
Hysterical	4	2
Conspicuous energy	8	4
Markedly ambitious	8	3
Schizophrenic		
somewhat	12	3
very	2	2
Cyclothymic		
somewhat	20	20
very	8	2

Neurotic symptoms

About one-third of the group had neurotic symptoms of various kinds and occurring in various degrees of severity. The symptoms and manifestations were of sufficient intensity to justify a diagnosis of neurosis in three patients, all of whom suffered from anxiety states. Table 2 gives the incidence of neurotic symptoms in the hay fever and control groups.

The hay fever group compared with the control group has a statistically higher incidence of symptoms of anxiety (38 per cent hay fever compared with 10 per cent controls, critical ratio = 3+) and tension (36 per cent hay fever compared with 8 per cent controls, critical ratio = 3+).

Emotional Tension and Stressful Life Situations in Hay Fever

The first attack of hay fever coincided with a period of emotional stress in 10 per cent of the group. This finding could, of course, be a chance association. It is

difficult to assess the probability of the onset of a disorder such as hay fever coinciding with stressful life situations purely by chance. In a control series of fifty herniotomy and appendicectomy patients of similar age and sex distribution as the hay fever patients it was found that the operation coincided with, or was immediately preceded by, a period of stress in 8 per cent. Thus the incidence is very similar in both groups,

TABLE 2.—NEUROTIC SYMPTOMS

Neurotic symptomatology	Hay fever group (%)		Control group (%)	
Anxiety				
mild	16		8	
moderate	12	38	2	10
severe	10		0	
Depression				
mild	10		4	
moderate	2	16	0	4
severe	4		0	
Tension				
mild	20		4	
moderate	8	36	4	8
severe	8		0	
Palpitations	26		6	
Dyspepsia	16		0	
Trembling	16		6	
Frequency of micturition	8		0	
Lassitude	18		0	

and there is no evidence that the incidence of stressful events is greater at the onset of hay fever than would be due to chance.

The following are examples of onset of hay fever coinciding with period of emotional stress:

Case (1). An adopted girl at the age of 18 discovered that she was illegitimate. She felt ashamed and humiliated but did not show her feelings and did not mention her discovery for some months. Her hay fever occurred in the spring about 2 months after this discovery.

Case (2). Developed her first attack of hay fever aged 22. She married at 21, her husband being killed in action some 6 months after the wedding. She was pregnant at the time and the baby was still born. Her hay fever developed during a period of marked grief.

Case (3). A female aged 25 developed hay fever the week before her wedding. Subsequently it was observed that emotional tension would bring on rhinorrhoea and lachrymation throughout the ensuing and subsequent hay fever seasons. Mention of the word "hay fever" precipitated hay fever symptoms, and she developed severe symptoms on receiving a letter inviting her to attend the Clinic for follow-up purposes.

Even though the association of stressful life situations with the onset of hay fever is not significantly greater than would be expected by chance, there is considerable clinical and experimental evidence indicating a possible causal relationship between the development of attacks of hay fever and emotional tension. Emotional changes may also exert summative effects with pollen allergy in producing hay fever symptoms. The association between the onset of hay fever and stressful life experiences may not

be irrelevant, as it is possible that the local tissue changes in the nose which occur during emotional states, may alter the threshold of reactivity to pollen, as will be described later. This may be a factor in determining the time of onset in these patients. Further support is given by the observation that in many of these subjects emotional tension subsequently precipitated attacks during the course of the stress.

TABLE 3

Personality traits	Emotional factors precipitating hay fever symptoms (%)	Emotional factors not precipitating hay fever symptoms (%)
General stability		
stable	86	54
moderately unstable	10	32
very unstable	3	14
Meek		
somewhat	23	59
very	20	23
Sensitive		
somewhat	26	23
very	20	63
Anxious		
somewhat	23	23
very	26	68
Meticulous or obsessional		
somewhat	29	40
very	20	45
Ambitious		
somewhat	16	45
very	13	0
Schizothymic		
somewhat	10	13
very	0	4·5
Cyclothymic		
somewhat	13	27
very	3	13·6

Influence of Emotions in Hay Fever Symptoms during the Course of the Illness

In 36 per cent of the group a close temporal association was noted between certain forms of emotional tension and the precipitation of hay fever nasal symptoms, viz. rhinorrhoea, sneezing. The following emotions were found to be capable of precipitating hay fever symptoms in these subjects: anxiety and tension, anger, resentment, feelings of humiliation and at times even pleasurable excitement. In some patients emotional tension resulting from working under pressure or against time appeared to be associated with the precipitation of attacks. The immediate development of symptoms with the above forms of emotional tension, together with the repeated evocation of hay fever symptoms by these emotions strongly suggests that the association is significant and relevant in precipitation of attacks.

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It is interesting to note that in some patients emotional changes will precipitate rhinorrhoea and sneezing only during the hay fever season, suggesting that the nasal mucosa is only affected by emotional changes when these operate in the presence of the pollen allergen or rather when the mucosa is in a state of dysfunction due to a pollen allergic reaction. Four patients developed asthma attacks with hay fever. In three of these the occurrence of asthma was at a period of emotional stress in the following cases:

Case 4. A man developed asthma with hay fever during a period of worry due to the ill health of wife and child.

Case 5. A woman, developed asthma during a prolonged stress relating to litigation over her mother's will and estate, the resulting strained relationships between the members of the family. Her hay fever consequently developed into a perennial rhinitis during this prolonged period of stress.

Case 6. A soldier, when suffering from hay fever symptoms, developed an attack of asthma when reprimanded by his Commanding Officer.

Conversion of seasonal hay fever rhinitis to perennial rhinitis

Some patients in the series who had a typical seasonal incidence of the nasal symptoms of hay fever over a period of years, subsequently developed nasal symptoms throughout the year. This conversion of seasonal rhinitis to perennial rhinitis in our group was considered to be due to three main factors:

(1) Some patients with typical hay fever later developed sensitivity to dust, feathers and other allergens, which precipitated attacks after the hay fever season and throughout the year.

(2) The superimposition of infections of nasal mucosa or nasal sinuses giving rise to perennial rhinitis.

(3) The effect of stressful life situations, with associated conflicts and emotional tension, on the autonomic nervous system controlling nasal mucosal functions.

Comparison of patients in whom emotional tension precipitated hay fever symptoms with those in whom emotional factors were unimportant

Do patients who have important psychological aspects to their hay fever, differ in personality or in neurosis from those in whom there is no evidence of significant psychological factors in the aetiology of their hay fever? From Table 3 it will be seen that the group in whom emotions precipitated hay fever symptoms had a higher proportion of persons showing instability of personality, marked timidity and over-anxiety. Table 4 shows that the incidence of anxiety, depression, tension, palpitations, dyspepsia and lassitude is significantly higher in the group in whom emotional tension precipitated hay fever symptoms.

Thus patients who have an emotional precipitation of hay fever symptoms are, in general, more unstable, have more neurotic traits of personality and a higher incidence of neurotic symptoms. These attributes have an important bearing on the patient's capacity to cope with stresses, and the tendency to develop emotional tension in response to various stressful life situations. The high incidence of anxiety and various somatic manifestation of emotional tension mediated by the autonomic nervous system may be particularly important in view of recent research indicating the important role played by the autonomic nervous system in allergic disorders (Rees, 1956).

Emotional factors in the nasal mucosal response to pollen

The association between emotional tension and the initial or subsequent attacks during the course of hay fever and the change from seasonal rhinitis to perennial rhinitis with emotional disturbances strongly suggests that emotional changes may influence the reactivity of the nasal mucosa to pollens.

TABLE 4

Neurotic symptomatology	Patients in whom emotional factors did not precipitate symptoms (%)		Patients with emotional precipitation of hay fever symptoms (%)	
Anxiety				
mild	11		24	
moderate	3·5	14·5	24	72
severe	0		24	
Depression				
mild	0		24	
moderate	0		45	37·5
severe	0		9	
Tension				
mild	17		24	
moderate	3·4	20·4	14	57
severe	0		19	
Palpitations	17		38	
Dyspepsia	3·5		34	
Trembling	10		24	
Frequency of micturition	3·5		14	
Lassitude	7		33	

Experiments by HOLMES, TREUTING and WOLFF (1951) have provided experimental validation for such clinical associations and a possible physiological basis for them. They studied the changes in the nasal mucosa under various conditions in a series of persons. The nasal mucous membrane was observed directly through a nasal speculum and the degree of swelling, secretion and blood flow changes were measured by standardized techniques.

Each subject was observed for 1 hr in a pollen room containing known concentration of mixed ragweed pollen kept circulating in the air by an electric fan. The exposure of both "non-sensitive" and "sensitive" individuals to mixed ragweed pollen elicited a response of hyperfunctioning characterized by hyperaemia, hypersecretion and swelling. When the nasal mucosa showed average function and the exposure to pollen was brief, the reaction of hyperfunctioning was well tolerated by the tissues of the nose. When the nasal mucosa was already hyperfunctioning, either as a result of emotional tension, infectious rhinitis or previous repeated exposure to atmospheric pollen, the additional nasal hyperfunctioning engendered, produced symptons and pathological tissue changes.

There is now convincing evidence that the nasal hyperfunctioning produced by emotional tension is mediated by parasympathetic neural pathways. Interruption of the sympathetic supply to the nose resulting from stellate ganglion block or surgical removal of the stellate ganglion gives rise to swelling of the nasal mucosa together with hyperaemia and hypersecretion (FOWLER, 1950; HOLMES, GOODELL and WOLFF, 1950). Later the hyperaemia may subside giving a swollen pale boggy mucous membrane. READING and MALCOMSON (1954) described a case in which section of the greater superficial petrosal nerve through a subtemporal craniotomy produced complete homolateral cessation of rhinorrhoea. The influence of the parasympathetic nervous system on the functions of the nasal mucosa provides an explanation for the precipitating role of emotions and for the interdependence and summative effects of emotional and allergic factors in hay fever.

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SUMMARY AND CONCLUSIONS

(1) A random sample of fifty hay fever patients attending an Allergy Clinic was studied from the physical and psychiatric aspects. A control group of fifty patients of corresponding age and sex distribution was also studied and assessed by identical methods.

(2) The age of onset of hay fever ranged from infancy to old age. Two-thirds of the series had the onset before the age of 25.

(3) There was no evidence to suggest a greater degree of neurotic predisposition in the hay fever group as a whole compared with the control group.

(4) No specific personality type was found in hay fever patients. 72 per cent had a stable and well adjusted personality. A variety of personality types were found among hay fever patients and while there was no specific personality pattern, certain trait ratings occurred in significantly high incidence, e.g. very anxious, very ambitious and very obsessional.

(5) No significant psychological factors were found in 64 per cent, emotional factors were considered to play a precipitating role in 36 per cent. In 10 per cent the onset of hay fever occurred during a period of severe emotional stress, and an additional 26 per cent, emotional tension precipitated hay fever symptoms during the course of the illness. In some patients the seasonal symptoms of hay fever became perennial during periods of stress with emotional changes capable of precipitating symptoms throughout the year. In the majority of patients whose rhinitis became perennial, emotional factors played an important role. Patients in whom hay fever symptoms were brought on by emotional changes differed from other hay fever subjects, in possessing a higher incidence of neurotic traits of personality, and of neurotic symptoms.

(6) A possible physiological basis in the tissue reaction and the probable role played by the autonomic nervous system in the interaction of emotional and allergic reactions are discussed.

(7) The results indicate that it is important to consider the hay fever patient as a whole, taking into account psychological, as well as physical aspects, so that the condition may be fully understood and adequately treated.

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ON THE MECHANISM OF THE DIURETIC CONDITIONED REFLEX*

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IF THE introduction of water into the organism is repeatedly linked with some stimulus, after a suitable number of associations the stimulus through the cerebral connections which developed alone without water intake will produce an increase in urine output: a diuretic conditioned reflex has been developed (BYKOV and BERGMAN, 1930; GROSSMAN, 1928; MARX, 1931; KOMMENDANTOVA, 1940). The role played by the system producing the antidiuretic hormone (ADH) in the efferent mechanism of the cerebral effect is shown by the experiments of BYKOV and ALEXEJEV-BERKMAN (1931) who found the conditioned reflex increase in diuresis unchanged after the denervation of the intact kidney. The efficiency of the *in situ* denervation may be questionable (QUINBY, 1916; HAMBURGER, 1936) but the experiments of BALAKSINA (1936) suggest that it might have affected the nervous pathway of regulation. These experiments showed that after injury to the infundibulum the conditioned effect, which is retained on the intact side, ceases in the denervated kidney. The humoral transfer is supported by the fact that LEBEGYEV and SZEVASZTJANOVA (1954) have elaborated a diuretic conditioned reflex in the dog kidney transplanted to the neck. However, the value of this interesting experiment is somewhat vitiated by the inconstancy of water and salt intake and by the small number of published data.

The involvement of a direct nervous action in the efferent mechanism of the conditioned response has not been definitely proved. In the experiments of BALAKSINA (1936) the temporary connections remain intact in both kidneys after the denervation of the unilateral one, but if the infundibulum is damaged, they disappear on the denervated side, but remain unaffected in the intact kidney. According to BALAKSINA (1936) this is because the cerebral cortical regulation of renal function has a double pathway. Following section of the pituitary stalk, the cerebral cortical impulses reach the innervated kidney through direct nervous pathways, but in the denervated organ both the nervous and the humoral transmission would cease. It is remarkable in these experiments that at a time when the denervated kidney does not respond to the cerebral cortical effect after the pituitary section, the diuresis occurring following loading with water remains completely unchanged. ADAM (1955) has observed after unilateral extirpation of the cerebral cortical motor and premotor areas a fall-out of conditioned reflexes in the kidney contralateral to the site of operation. However, the conditioned reflexes described in his experiments are very few, and it is therefore difficult to decide how this unilateral fall-out of conditioned reflexes can be brought into line with experiments concerning the role of the humoral mechanism.

In our own experiments we have approached the efferent mechanism of the cerebral cortical effect by studies on the volume and composition of excreted urine.

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METHODS

First, the characteristics of the unconditioned reflex water diuresis following water intake in 6 dogs was determined in 118 experiments without water loading, and in 480 experiments involving the intake of 400 ml of water. The conditioned diuretic reflex was then elaborated in 2 dogs, in 233 experiments, on 41 occasions.

Female dogs with ureteral fistulae, weighing 12 to 25 kg were used. After fasting for 16–18 hr and thirsting for 10–14 hr, the same experimenter placed the animals at the same time every day on the Pavlov stand in a soundproof reflex chamber and collected the urine every 15 min. The urine was tested for volume, endogenous creatinine and sodium content, as well as for osmolar concentration. After it was found that there were only minor variations, serum creatinine and sodium levels were estimated at 2 week intervals. The methods and procedures involved have been described elsewhere (GERBNER, KOVACS and ALTMAN, 1959. In press).

Apart from the environment, the conditioned stimulus was a generator sound applied 30 sec before the ingestion of water. The differentiating stimulus was a different room, in which the animals were given no water to drink. The unconditioned stimulus was 400 ml water flavoured with 5 g of powdered milk, which the animals drank spontaneously. In the bitch Kincses, a conditioned reflex was elaborated, then differentiation was effected in the other room, and finally the positive conditioned reflex was extinguished. In the bitch Füles the elaborated conditioned diuretic reflex was extinguished; then a second conditioned reflex was elaborated to another generator sound and finally this, too, was extinguished. In six experiments the completion of the conditioned reflex elaborated by the usual method was not followed by a transfer back to the cage, but the animals were given 400 ml of water to drink. Thus, while during the first two hours of the experiment the conditioned reflex diuresis was obtained, the most important conditioned stimuli (leading the animal to the experiment, placing on to the Pavlov stand, conditioned sound stimulus) were absent and a regular unconditioned water diuresis resulted, during the second 2 hr.

Because of the variations in urine output (GERBNER and KOVACS, 1959. In press) we accepted as an increase in conditioned reflex diuresis only that, which showed a regular curve after the conditioned stimulus (without water intake), the maximum minute diuresis of which increased to at least 3 times the control value and in which during the 2 hr period following the conditioned stimulus, at least the double of the no-water intake control urine volume is voided (this exceeding the $2s = 2\sqrt{[\varepsilon(x - \bar{x})^2/(n - 1)]}$ mathematical scattering).

RESULTS

In the control experiments without water load, diuresis and sodium excretion are low and steady, the GFR*, as measured by the endogenous creatinine clearance method, shows little change, the urine is concentrated and the concentration is nearly constant.

After the intake of water the volume of urine output markedly increases, reaching the maximum usually 1 hr after drinking, then beginning to decline. As diuresis is increasing, GFR, too, increases, reaches a peak before that for diuresis, then declines gradually. The urine turns hyposmotic, then begins to increase in concentration. Sodium excretion (before the conditioned reflex is established) is low and almost constant (Fig. 1).

The conditioned reflex diuresis usually starts from a level higher than the control one and reaches its peak sooner than does the water diuresis. Maximum minute diuresis increases more than once to 8 to 10 times the no-water-intake control value, the total amount of urine output (during the 2 hr of the experiment) to 5 to 6 times higher than with water diuresis. As diuresis is increasing, GFR slightly increases, then gradually decreases. The concentration of the urine decreases, then increases, but the degree of dilution is smaller than that seen in water diuresis, in spite of the significant increase in volume. The urine excreted as a response to the conditioned effect becomes hypotonic only infrequently, at very high diuresis levels. The free

* Glomerular filtration rate.

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	Unconditioned diuretic reflex								Conditioned diuretic reflex							
	1	2	3	4	5	6	7	8	1	2	3	4	5	6	7	8
V (ml)	0·62	1·78	4·11	4·93	5·45	4·03	2·38	1·57	4·11	4·28	2·96	2·43	1·52	1·04	0·87	0·73
U_{cr} (mg %)	137	70	29	22	18	21	31	42	20	20	23	27	36	53	62	75
U_{Na} (μequiv/l.)	96	55	25	17	13	13	24	25	75	63	67	78	112	128	139	145
U_{osm} (cmosm/l.)	850	448	178	135	124	156	226	388	350	301	344	397	565	753	834	990
E_{cr} (g^{-5})	85	124	119	108	98	85	74	66	82	86	68	66	55	55	54	55
GFR	50	85	81	74	67	58	50	45	73	76	61	59	49	49	48	49
E_{Na} (μequiv)	60	98	103	84	71	52	57	39	308	270	198	190	170	133	121	106
Na (%)	0·7	0·7	0·8	0·7	0·7	0·6	0·7	0·6	2·6	2·2	2·0	2·0	2·2	1·7	1·6	1·3

Serum creatin 1·47 mg% Serum Na 154 μequiv/l. Serum creatin 112 mg% Serum Na 161 μequiv/l.

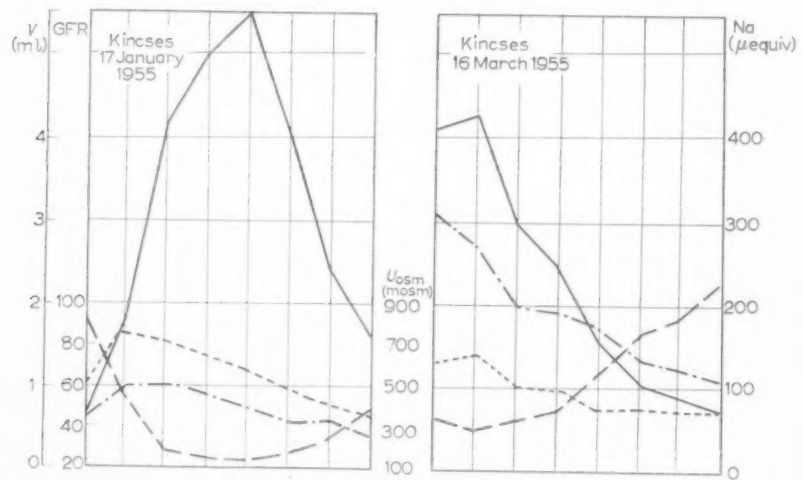

FIG. 1.—Unconditioned water diuresis and conditioned duretic reflex. (*Below*) the curves. (*Above*) the numerical values the curves were plotted from (computed for square metre of body surface).

Ordinates: $V_{(ml)}$: minute diuresis; $Na_{(\mu equiv)}$ and $E_{Na(\mu equiv)}$: sodium excretion, GFR: glomerular filtration rate, E_{cr} (g^{-5}): creatinine excretion, U_{kr} (mg%): creatinine concentration, U_{Na}(μequiv/l.): sodium concentration; U_{osm}cmosm/l.: osmolar concentration; Na(%): excreted Na in percentage of filtrated Na.

Abscissae (1-8): 15 min periods. ———: diuresis; . . . . . : filtration; – – – – – – –: osmotic concentration; — · — · — · — : sodium excretion.

water clearance thus becomes even more strongly negative in smaller conditioned reflexes and turns slightly positive only with much diuresis.

The most remarkable feature is the extreme increase in natriuresis, that may amount to more than 8 times the values for water diuresis and for the no-water-intake experiments at the peak of conditioned diuresis. The increase is very substantial as compared to the amount of sodium filtrated, as well as in the total diuresis during the entire experiment (Fig. 1).

The characteristics of the conditioned reflex diuresis: the increase in urine output, the slight increase in GFR, the moderate decrease in osmotic concentration and the marked increase in natriuresis disappear when the conditioned reflex is inhibited

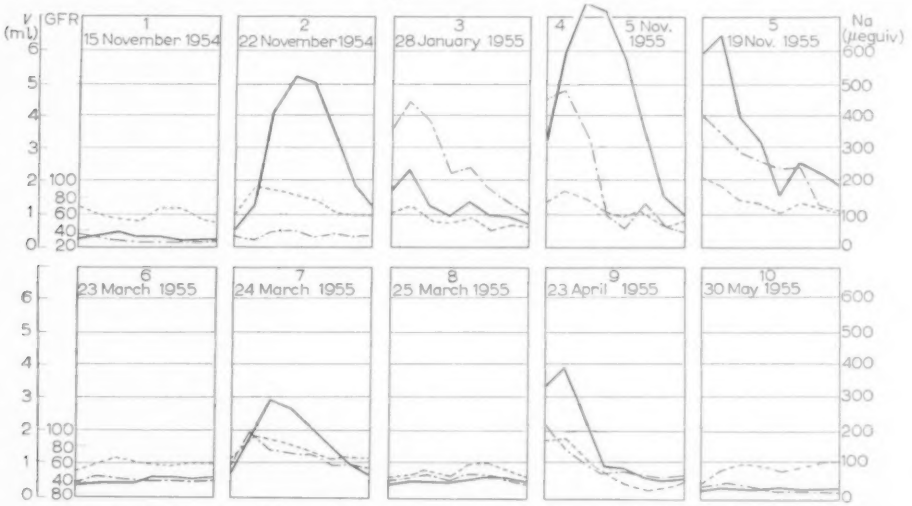

FIG. 2.—Elaboration, differentiation and extinction of the conditioned diuretic reflex. The data are given for square metre body surface.
(1) Control without water load; spontaneous diuresis. (2) Unconditioned water diuresis (reinforcement) before establishment of conditioned reflex. (3) Conditioned diuretic reflex. (4) Unconditioned water diuresis (reinforcement) after establishment of conditioned reflex. (5) Conditioned diuretic reflex. (6) Differentiation in "differentiating" room; spontaneous diuresis. (7) Conditioned reflex in "active" room. (8) Differentiation in "differentiating" room; spontaneous diuresis. (9) Conditioned reflex in "active" room. (10) Extinction in "active" room; spontaneous diuresis.
Ordinates: V(ml)- minute diuresis; Na(μ equiv.): sodium excretion; GFR: glomerular filtration rate; ——— : diuresis; . . . . . : filtration; —·—·—·— : sodium excretion.

(in the course of differentiation and extinction), and reappear when it is restored (during differentiation as a result of exposure to positive conditioned stimuli, as well as on the repeated elaboration of the conditioned reflex) (Fig. 2).

When we produced within the same experiment the conditioned diuretic reflex and the unconditioned reflex water diuresis, the two patterns described above were seen within the same experiment. In the conditioned reflex increase in diuresis, an increase in filtration, a slight decrease in concentration of the urine, and a high natriuresis were seen, whereas, in the unconditioned water diuresis, a greater increase in filtration, a significant reduction in the concentration of the urine and a low natriuresis were observed (Fig. 3).

In the experiments, in which the established conditioned reflex was further reinforced, i.e. both the conditioned stimulus and water loading were used, the

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	Conditioned diuretic reflex								Unconditioned diuretic reflex							
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
V (ml/m)	0·51	1·22	2·59	2·97	1·28	1·06	1·43	0·86	0·96	2·99	5·49	5·49	3·32	1·78	1·64	1·00
U_{cr} (mg %)	93	63	62	45	52	62	62	71	78	40	29	18	19	26	30	44
U_{Na} (μequiv/l.)	340	222	456	448	114	127	109	103	101	52	24	13	14	16	19	21
U_{osm} (mosm/l.)	1076	781	587	468	468	609	538	619	694	425	240	118	145	231	301	366
E_{cr} (g^{-5})	47	77	135	134	95	65	89	61	75	119	159	99	63	47	49	44
GFR	40	65	113	113	79	55	74	51	63	100	134	83	53	39	41	37
E_{Na} (μequiv/m)	158	271	404	350	208	135	156	89	97	155	132	71	46	28	31	21
Na (%)	2·6	2·8	2·4	2·1	1·7	1·6	1·4	1·2	1·0	1·0	0·7	0·6	0·6	0·5	0·5	0·4

Serum creatin 1·19 mg % Serum Na 151 μequiv/l.

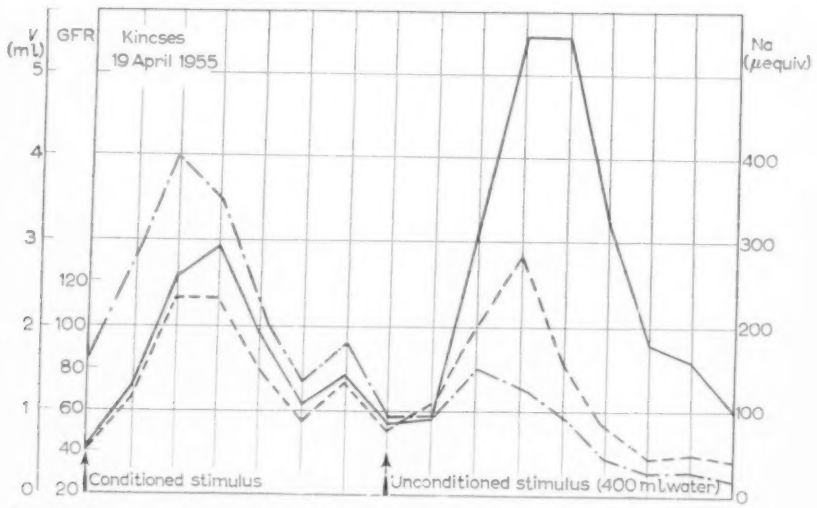

FIG. 3.—Conditioned and unconditioned diuretic reflex within the same experiment. The arrows indicate the application of the conditioned and of the unconditioned (400 ml) of water stimulus. Abbreviations and signs as in Fig. 1.

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TABLE 1.—BITCH KINCSES (25 kg)

	(1) Means and scatterings for each group			
	V	Na	Kr	Osm
(A)	34·9 ± 12·2	3·7 ± 1·5	72·5 ±16·5	48·1 ± 8·3
(B)	334·5 ± 44·7	6·8 ± 1·6	87·9 ±11·7	74·3 ±16·5
(C)	143·8 ± 51·4	15 ± 5·5	79·3 ± 7·4	91·6 ±22·1
(D)	362·8 ±103·9	11·5 ± 4·5	83·9 ±14·8	95·9 ±16·6

	(2) Significance between single groups (t test)			
	V	Na	Kr	Osm
(A–B)	$p < 0{\cdot}01$	$p < 0{\cdot}01$	$p < 0{\cdot}01$	$p < 0{\cdot}01$
(A–C)	$p < 0{\cdot}01$	$p < 0{\cdot}01$	$0{\cdot}05 < p < 0{\cdot}10$	$p < 0{\cdot}01$
(A–D)	$p < 0{\cdot}01$	$p < 0{\cdot}01$	$0{\cdot}02 < p < 0{\cdot}05$	$p < 0{\cdot}01$
(B–C)	$p < 0{\cdot}01$	$p < 0{\cdot}01$	$0{\cdot}02 < p < 0{\cdot}05$	$0{\cdot}01 < p < 0{\cdot}02$
(B–D)	$0{\cdot}2 < p < 0{\cdot}50$	$p < 0{\cdot}01$	$0{\cdot}2 < p < 0{\cdot}50$	$p < 0{\cdot}01$
(C–D)	$p < 0{\cdot}01$	$0{\cdot}02 < p < 0{\cdot}05$	$0{\cdot}10 < p < 0{\cdot}20$	$0{\cdot}20 < p < 0{\cdot}50$

(A) Control without water load
(B) Control with water load
(C) Conditioned reflex experiments
(D) Reinforcement while conditioned reflex still exists

V : urine output (ml/120 min).
Na : sodium excretion. mE/120 min.
Kr : creatinine excretion, mg/120 min.
Osm : osmolar excretion mosm/120 min.

The number of experiments (n) is 25 in each group.

unconditioned reflex water diuresis also changed in its pattern. As in the case of the conditioned reflex, the diuresis curve usually starts from a higher level, reaches a higher peak sooner than in the water diuresis preceding the elaboration of the conditioned reflex and the natriuresis is also significantly higher (Figs. 2, graphs 2 and 4, and Table 1).

DISCUSSION

The conditioned reflex diuresis is presumably high at the beginning of the experiment because the leading of the animal into the experimental room, and its placing on the stand are also parts of the conditioned stimulus complex and thus start an increase in water diuresis. This is in connection with the mode of the formation of temporary connections with the biological role of conditioned reflexes.

The increase in filtration observable in the conditioned diuretic reflex (usually smaller than the unconditioned water diuresis) suggests that the glomerular change may be a part of the mechanism of the diuretic conditioned reflex. However, the significant increase in the unconditioned water diuresis, is not statistically significant

in the conditioned reflex and, owing to the methodical difficulties of GFR estimations (GERBNER, KOVACS and ALTMAN. In press) is less certain. On the other hand, the increase in the dilution of the urine (even if only slight) indicates a reduction in the tubular reabsorption of water, which, in turn, indicates that the production of ADH has diminished.

The high increase in natriuresis is due to the fact that whereas the volume of urine significantly increases, its sodium (and osmolar) concentration decreases relatively slightly. The changes in the free water clearance point in the same direction. As

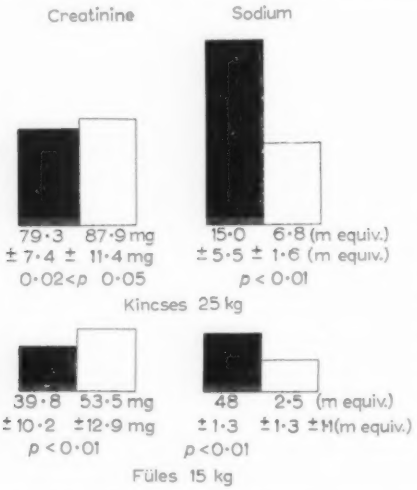

FIG. 4.—Comparison of the creatinine and sodium excretion in the conditioned and in the unconditioned diuretic reflex. The solid columns show the values excreted in the conditioned, the empty ones those obtained in the unconditioned reflex experiments. On the basis of 25 unconditioned and 25 conditioned reflex experiments made on Kincses and 9 conditioned and 25 unconditioned reflex experiments on Füles.

in the conditioned diuresis substantially more sodium is excreted at a low GFR than at the higher GFR of the unconditioned water diuresis, the increased natriuresis of the conditioned diuretic reflex is the result not of an increase in GFR, but chiefly of a reduction in the tubular sodium reabsorption (Fig. 4).

This is particularly obvious in the cases in which the conditioned diuretic reflex and the unconditioned water diuresis were elicited within the same experiment. In the first half of the experiment, under the influence of conditioned stimuli there were many periods in which at lower filtration more sodium was excreted than at the higher filtration of the unconditioned water diuresis resulting in the second half. Thus, the cerebral cortex (where conditioned reflexes are formed) can influence tubular sodium reabsorption independent of filtration.

In the reinforcements following the establishment of the conditioned reflex, the conditioned stimuli are used just as in the conditioned reflex experiments without reinforcement. It is probable that in these the conditioned and the unconditioned effects are summated, and this results in the increase in the diuresis curve, and in the enhanced natriuresis.

Thus, according to our theory concerning the mechanism of conditioned reflexes, after the establishment of the temporary connection the impulse generated by the conditioned stimulus reaches the centre of the unconditioned reflex and elicits through its effector mechanism the unconditioned effect (MAJOROV, 1954; ASZTRATIAN, 1955). If the effector mechanism were the same in both cases, the composition of the urine would not differ in the conditioned reflex water diuresis from that in the unconditioned

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water diuresis. However, with its increased natriuresis the conditioned diuretic reflex deviates from the mechanism of that unconditioned reflex, to which it had been elaborated. Tubular sodium reabsorption, as it occurs in diuresis due to the temporary connections, is different from that in the after-drinking unconditioned water diuresis. This cannot be explained by former theory. For this reason it is to be assumed that other factors are also involved in the mechanism of temporary connections.

SUMMARY

The conditioned diuretic reflex is more rapid in its course than is the unconditioned water diuresis produced by water intake. In the mechanism of its development the slight increase in filtration is not significant and does not prove a glomerular effect. The concentration of the urine decreases, which indicates that the secretion of the antidiuretic hormone has diminished.

In spite of the significant increase in the volume of the urine, the decrease in its concentration is smaller than that which is observed in the unconditioned water diuresis. As a result of this, in the diuretic conditioned reflex the sodium and the osmolar excretion are substantially higher in value than are the values for the experiments without water intake and for the unconditioned water diuresis. This increase in natriuresis is due, not to an increase in filtration, but primarily to a diminution in tubular sodium reabsorption. Thus, the cerebral cortex influences tubular sodium reabsorption independent of filtration.

The known theory of the conditioned reflex, according to which the impulse reaches the centre of the unconditioned reflex and elicits through its effector mechanism the conditioned effect, does not explain the difference in the mechanism of the conditioned reflex and the unconditioned water diuresis. It is to be assumed that other factors are also involved in the mechanism of the temporary connections.

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REPORT OF MEETINGS

SOCIÉTÉ FRANÇAISE DE MÉDECINE PSYCHOSOMATIQUE

Comte rendu réunion du Jeudi 27 Février 1958

R. HELD: *A propos d'accidents d'automobile quelques notes de clinique psychanalytique*

Le matériel recueilli au cours de séances de psychanalyse-type ou de psychothérapies permet une étude dynamique de la psychopathologie de beaucoup de conducteurs d'automobile ayant eu ou provoqué des accidents ou susceptibles d'en provoquer. Les aspects symboliques de la conduite des automobiles ou de l'automobile elle-même sont envisagés (puissance, narcissisme, extension du Moi du propriétaire de l'auto au véhicule, etc.). Quelques rêves significatifs et quelques cas cliniques sont rapportés. Le rôle de l'agressivité, du sado-masochisme; les complexes analytiques de base (Oedipe, castration) sont mis en évidence. Référence particulière est fait à *l'oralite* ainsi qu'aux comportements masochiques on rapport avec des sentiments inconscients de culpabilité. Un interrogatoire-type du conducteur accidenté est proposé. Après comparaison des accidents d'automobile et du travail, l'auteur, très schématiquement, conclut à la primauté du masochisme en matière d'accidents du travail, à celle du sadisme en matière d'accidents d'automobile. Toutefois cette dichotomie n'a rien d'absolu.

V. GACHKEL et C. LEROY: *Psoriasis familial à déclenchement émotionnel chez trois personnes*

Observation de deux soeurs, jeunes femmes de 16 et 25 ans et de la fille de cette dernière âgée de 6 ans.

Chez les trois sujets un conflit familial intense déclenche pour des raisons différentes des poussées de psoriasis après un délai de 48 hr du traumatisme affectif.

L'isolement ou la résolution du conflit guérit la poussée en quelques jours, une récidive des problèmes le fait réapparaître. Il ne semble pas que la localisation cutanée du psoriasis acquière ici une expression symbolique particulière d'un conflit déterminé.

Un groupage sanguin des six personnes de la famille n'amène pas d'éléments nouveaux.

Il semble bien exister chez les trois sujets une "disposition à faire du psoriaris" pour des conflits tout à fait différents chez les soeurs et chez la petite fille.

C. BRISSET: *L'Angoisse comme expérience psychosomatique fondamentale*

L'angoisse est en elle-même une expérience psychosomatique vécue comme désarroi de la conscience et comme expression émotionelle aigüe. Si la panique, ou peur suraigüe, pour être considérée comme une "réaction" aux évènements, l'angoisse commune ne peut être comprise que comme ayant une origine interne: c'est un conflit de tendances. A ce titre, elle est l'expérience que les psychoses tentent de nier, que les pervers refusent systématiquement, que les névrosés évitent par des conduites

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de "rétrécissement" du champ de la conscience. Quant aux malades psychosomatiques, leur comportement consiste à nier l'angoisse on tant qu'expérience vécue, ce qui ouvre un champ plus vaste à l'angoisse on tant que manifestation corporelle.

On peut dire que dans cette vus du problème le psychosomatique participe des conduites d'exclusion d'une partie de l'expérience humaine. Il convient de distinguer les manifestations psychosomatiques vraies qui répondent à cette définition et qui sont des maladies organisées tout au long d'une vie de par la prévalence de réponses toujours les mêmes aux conditions de l'angoisse, et les manifestations psychosomatiques passagères qui répondent au cadre de l'ancienne hystérie: ce sont des symptômes qui excluent momentanément l'angoisse par une expression corporelle symbolique reversible et n'atteignant généralement pas le niveau cellulaire ou tissulaire à cause de leur durée courte ou relativement court.

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Papers to be published in future issues.

LINFORD REES: An evaluation of the role of emotional factors in miner's nystagmus.

D. WALTON and D. A. BLACK: The application of modern learning theory to the treatment of chronic hysterical aphonia.

M. GERBNER, M. B. KOVACS and K. ALTMAN: On the glomerular factor in water diuresis.

M. GERBNER, M. B. KOVACS and K. ALTMAN: The effect of adjustment to the environment and its alteration with water diuresis.

CHARLES SHAGASS, KARLA MULLER and B. ACOSTA: The pentothal sleep threshold as an indicator of effective change.

BRIAN ACKNER and G. PAMPIGLIONE: An evaluation of the sedation threshold rates.

J. L. TITCHENER, M. B. SHELDON and M. D. ROSS: Changes in blood pressure in hypertensive patients with and without group psychotherapy.

M. GERBNER and M. B. KOVACS: Effect of stimuli of variable intensity on urine output in unanaesthetized dogs.

T. D. TJOSSEM, A. R. LEIDER, R. W. DEISHER, T. H. HOLMES and H. S. RIPLEY: Emotional reactions and skin temperature responses in children aged two to four years.

P. V. CARDON and R. S. GORDON: Rapid increase in plasma unesterified fatty acids in man during fear.

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THE PENTOTHAL "SLEEP" THRESHOLD AS AN INDICATOR OF AFFECTIVE CHANGE*†

C. SHAGASS, M.D., KARLA MULLER, M.D. and H. B. ACOSTA, M.D.

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EXTENSIVE studies of psychiatric patients with the sedation threshold have demonstrated that this index of sedative tolerance is related to several variables of psychiatric interest (SHAGASS, 1956; SHAGASS and JONES, 1958). Recently, a modification of the sedation threshold, which is much simpler because it does not involve measuring the EEG, was described and validated (SHAGASS and KERENYI, 1958). This was called the "sleep" threshold; it represents the amount of amobarbital required to produce unresponsiveness to verbal stimulation. The amobarbital "sleep" threshold was shown to be correlated with the sedation threshold (r, 0·65), and to provide almost the same diagnostic differentiation between patients with neurotic and psychotic depressions. Because of the relative ease with which "sleep" thresholds can be determined, the procedure may be applied under conditions which would make testing of the EEG-determined sedation threshold difficult or impossible.

This study presents the results obtained with a further modification of the "sleep" threshold, using thiopental sodium (*pentothal*). The details of this modification were governed by practical considerations related to the aims of the study. These aims were as follows:

(1) To determine the repeatability of the "sleep" threshold when individuals are tested on numerous occasions.

(2) To determine the effect of electroconvulsive therapy (ECT) on the "sleep" threshold.

(3) To determine whether affective changes, or reactions, will be reflected in "sleep" threshold changes.

As the administration of a course of ECT provides the opportunity to give multiple pentothal injections for purposes of premedication, and as ECT generally produces marked affective changes in a relatively brief time, this treatment situation seemed ideal for present aims. By making the premedication into a "sleep" threshold test, repeated determinations could be carried out and the results correlated with clinical observations. A special interest of the study was in connexion with affective reactions to the treatment itself, such as fears before commencement of treatment and so-called ECT anxiety, which occurs as a complication after several treatments (ALEXANDER, 1953). It was also hoped that the data might suggest applications of the "sleep" threshold as an indicator for judging therapeutic progress with ECT.

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METHODS AND MATERIALS

"Sleep" threshold procedure

Atropine, 0·6 mg, was injected subcutaneously approximately 30 min before every "sleep" threshold determination, as this is part of the routine ECT premedication. For the test, pentothal was diluted with water so that 1 ml contained 0·5 mg/kg body weight; 1 ml was injected intravenously every 30 sec. The patient was instructed beforehand in the following manner: "I am going to give you an injection to put you to sleep. Once in a while, I am going to call your name, and I want you to let me know whether you can still hear me. If you can, say 'yes', or do something to let me know. I want to be sure you are asleep." Obviously the usual patient would be most eager to be completely unconscious before treatment. Before the first test which was not to be followed by treatment the instructions were preceded by the statement: "You are to receive a treatment tomorrow (or later today). Today we will do a test to find out how much sedative you will need, so that we can be sure you will be asleep for the treatment. This is only a test; you will not get a treatment now". The second non-treatment test was explained to the patient as an attempt to find out whether his sedative needs had changed. The doctor giving the injection talked to the patient during the procedure; shortly before the next ml was to be injected, the patient's name was called. Any verbal, or apparently voluntary motor response, was accepted as a sign that the patient was still "awake", and the injection was continued. The usual motor responses consisted of: opening eyes, nodding the head, head turning in direction of examiner, movement of lips (probably in an attempt to verbalize).

The "sleep" threshold was the amount of pentothal (mg/kg) required to reach the point when there was neither a verbal nor motor response to calling the patient's name. Some difficulty was occasionally encountered in distinguishing the point of unresponsiveness when there was an automatic reaction, such as eye blinking, in response to the verbal stimulus. Although there was always the possibility that absence of response might occur because the patient deliberately refused to co-operate, this was apparently not encountered. Such a situation could probably be dealt with by applying a painful stimulus, e.g. supra-orbital pressure, to elicit a voluntary reaction. However, in most instances, the simple criterion of absence of verbal or motor response to verbal stimulation seems adequate.

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Subjects

Twenty-eight patients were studied. There were twelve men and sixteen women ranging in age from 16 to 67, with a median age of 46 years. Diagnoses were: psychotic depression, 8; neurotic depression, 7; schizophrenia, 11; paranoid state, 1; manic psychosis, 1. Tests were conducted over the full course of treatment in nineteen of the twenty-eight patients. For one patient the last treatment was missed, one had to be placed on special precautions after his first treatment, and one left hospital after receiving two treatments. In the remaining six cases the tests were not continued to the end of treatment because the treatment courses were very long and maintenance therapy was instituted. At least six treatment tests were obtained in twenty-six patients.

Sequence of tests

The initial aim was to obtain at least two "sleep" threshold determinations on each patient outside of the ECT situation. One test was done before any treatments were administered, usually on the day preceding the first treatment, but sometimes on the same day at least 4 hr before the first treatment. Although it was planned to carry out a second test on the day after the first treatment, this was frequently not possible. Only nineteen second tests were obtained and, of these, six were not carried out until after the second treatment, usually because the patient was put on daily treatments.

Clinical data

The doctor administering the injection recorded any noticeable reactions or comments of the patient during the non-treatment tests and the initial treatments. A second doctor, who carried out most of the actual treatment tests, was instructed to record any special observations made during treatments; such data, however, seemed very meagre in volume and may be regarded as inadequately recorded, probably because of the rush of treatment routine. In analysis of clinical correlates the nurses' routine ward notes were used as the main source of clinical data. The senior investigator abstracted the nurses' notes, following the system of writing down opposite the date of each test the descriptive phrases used by the nurses in the interval between the preceding test and this one. He also wrote in any notes or comments on behaviour made by the doctors administering the tests.

The sheets containing abstracts of clinical observations, mainly by the nurses, were then independently classified by two of the investigators. The following categories, which reflect hypotheses as to the type of relationships possibly present, were used in classification: no apparent clinical change; fear of treatment expressed for the first time (i.e. coded only when first observed and recorded); improvement of behaviour and feeling state ("better"); increased symptoms, dysphoria or disturbed behaviour ("worse"); disorientation; increased drowsiness; improvement considered to be the beginning of a remission; "special" events; tests following "special" events. The "special" events category will be described more fully in the results section. These clinical categories were then compared with the measured changes in the "sleep" threshold.

RESULTS

Reliability of judging unresponsiveness

To determine the extent of variation between observers in judging unresponsiveness, thirteen tests were carried out in which three to five observers, including the doctor giving the injection, independently judged the onset of unresponsiveness.

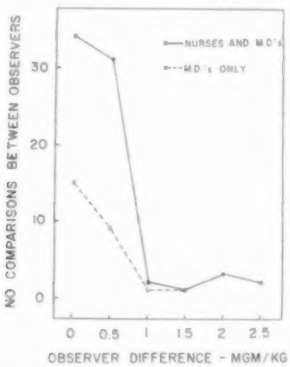

FIG. 1.—Distribution of differences between observers in judging unresponsiveness.

The injection was continued for several ml after the doctor giving the injection considered the threshold to have been reached. The other observers noted the time elapsed between the first injection and the time when they considered the patient unresponsive. By using each possible pair of observers, there were seventy-three inter-observer comparisons. It was also considered of interest to determine the agreement between doctors and nurses separately. The data plotted in Fig. 1 show the incidence of deviations between observers. For all observers, sixty-five out of seventy-three pairs of comparisons did not deviate from one another by more than 0·5 mg/kg. For doctors alone, twenty-four of twenty-six comparisons were within 0·5 mg/kg. With one exception the deviations greater than 0·5 mg/kg occurred in two tests. In one of these it seems that the nurses were taking, as a sign of responsiveness, a reaction considered by the doctors to be involuntary random movement. It thus appeared that the probable error of the test determination, if properly carried out, should be no greater than 0·5 mg/kg.

Test–retest reliability

The repeated testing of individuals, usually over ten times each, provided an opportunity to assess the degree of stability of the pentothal "sleep" threshold under the conditions of this study. Because of the varying changes produced by ECT,

these conditions may safely be regarded as less than ideal for maintenance of a stable measurement. Consequently present data reflect less than optimal reliability. Rank order correlation coefficients between successive tests were computed for all available cases until the number was too small for any meaningful determination. Coefficients were also calculated for a group of nineteen patients, all of whom had complete testing for at least eight treatments. The correlation coefficients are shown in Table 1.

TABLE 1.—CORRELATIONS BETWEEN SUCCESSIVE TESTS (RANK ORDER COEFFICIENTS)

Tests	All available cases		19 Cases with at least 8 ECT
	r_1	No. of cases	
0^1–0^2	0·77	19	0·81
0^1–1	0·57	28	0·77
1–2	0·76	27	0·82
2–3	0·68	25	0·65
3–4	0·73	25	0·61
4–5	0·87	24	0·86
5–6	0·80	24	0·78
6–7	0·65	21	0·59
7–8	0·78	20	0·77
8–9	0·81	16	
9–10	0·90	12	

It will be noted that the lowest coefficient was 0·57. It was obtained between the pre-treatment test and the test done in conjunction with the first treatment for the group as a whole. This probably represents the greatest change in conditions of testing. The other coefficients ranged from 0·59 to 0·90 with fourteen of twenty above 0·70. All of the coefficients in Table 1 were significant at the 1 per cent level of confidence. These results indicated that the pentothal "sleep" threshold has an acceptable degree of test–retest reliability, but that there was also a not inconsiderable degree of deviation from one test to another. The extent to which known emotional factors may account for the variability of the threshold will be considered subsequently.

Effect of ECT on the "sleep" threshold

Because of the variable number of treatments received by each patient, two methods for assessing the effect of ECT on the "sleep" threshold were employed. In one, the data for those nineteen patients who had had complete testing for at least eight treatments, were studied for those eight treatments. The bottom part of Fig. 2 shows the mean "sleep" threshold values for the pre-treatment test and for each of the eight treatments in this group. It will be seen that, on the whole, there was a tendency for the "sleep" threshold to increase as the course of treatments progressed. The statistical validity of this conclusion was determined by carrying out an analysis of variance. It was found that comparison of the variance between tests with the residual, after the variance between individuals had been removed, resulted in a value of 3·01 for the *F*-ratio, significant at the 1 per cent level of confidence.

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Another method of demonstrating the average rise of the "sleep" threshold with ECT was to divide the tests available for each case into thirds. Thus, if a patient received twelve treatments, each third would contain four tests; whereas patients who received only six treatments would have only two tests in each third. For the twenty-six patients who had had six or more treatments, the mean threshold value increased from 3·71 in the first third of treatments to 4·22 mg/kg in the last third of

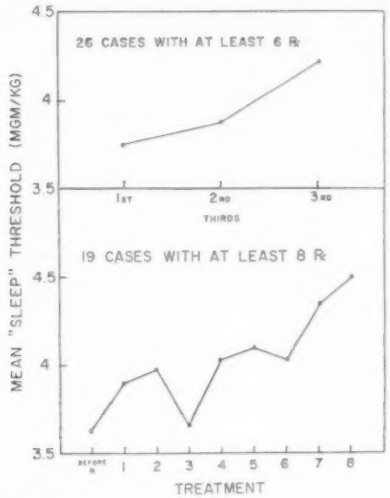

FIG. 2.—Mean "sleep" thresholds with progress of ECT. Upper curve shows means of successive thirds of available tests in twenty-six cases with at least six treatments. Lower curve shows successive means in nineteen cases with complete data for at least eight treatments.

treatments. This difference was significant at the 1 per cent level of confidence. The mean threshold values for successive thirds of treatments are shown in the top half of Fig. 2.

It may therefore be concluded that, on the average, ECT produces a rise in the "sleep" threshold. One possible objection to this conclusion could be raised on the ground that, with repeated administration of pentothal, tolerance to this agent may develop and be reflected in a higher "sleep" threshold. Although this possibility cannot be completely ruled out on the basis of the present data, it may be pointed out that the rise in "sleep" threshold occurred in only seventeen of the twenty-six patients, and that the remainder showed slight decreases from the first to the last third of treatments. Furthermore, one patient who was studied for twenty treatments and two patients studied for eleven treatments showed a decrease.

An attempt was made to determine whether the change in "sleep" threshold was related to the therapeutic response to ECT. The response to ECT in twenty-four cases was classified as: marked, moderate, and slight or none, on the basis of opinions expressed by the treating physicians. No significant relationship was found between quality of therapeutic effect and the amount of shift in the "sleep" threshold from the first to the last third of tests.

As there seemed to be some possible relationship between the number of treatments for which tests were carried out and the amount of change between the initial and last

third of tests, the correlation between these two variables was determined. The rank order coefficient was 0·417 which was significant at the 5 per cent level of confidence. The magnitude of the change from first to last third was not correlated with the mean value for the initial third of tests, or the pre-treatment threshold; the rank order coefficients were —0·01 and —0·05 respectively. It would appear that, of the factors investigated, only the number of treatments was related to the increase of "sleep" threshold with ECT.

Relationship between initial "sleep" threshold and diagnosis

The previous study with the amobarbital "sleep" threshold indicated that the "sleep" threshold may provide correlations with diagnosis similar to those obtained with the sedation threshold. It was consequently a matter of interest to determine what relationships existed in the present series, even though the numbers were small.

Table 2.—Distributions of pre-treatment "sleep" thresholds in relation to prediction from diagnosis and to outcome with ECT

"Sleep" threshold (mg/kg)	Predicted threshold		Improvement with ECT			
	High	Low	Marked	Moderate	Slight or none	Uncertain
1·5		1				1
2·0		2	1			
2·5	1	6	4	1		1
3·0		4	2	2	1	
3·5	1	1	1	1	2	
4·0	1	1			1	
4·5	2	2			1	2
5·0	2			2	1	
5·5	2		1		1	
6·0	1			1		
8·0	1				1	
	Total 11	17	9	7	8	4
3 or less	1	13	7	3	1	
3·5 or more	10	4	2	4	7	

The pre-treatment "sleep" threshold was used for this purpose, as it would correspond to a diagnostic test. The patients were classified into the following groups: psychotic depression (8), paranoid state (1), manic psychosis (1), acute or "acute on chronic" schizophrenia (7), chronic schizophrenia (4), neurotic depression (7). According to previous experience with the sedation threshold, one would expect a high threshold in patients with neurotic depression and chronic schizophrenia and a low threshold in the other patient groups. The distributions of the thresholds in the groups predicted to have high and low values are shown in Table 2. Also shown are the distributions of the thresholds in those cases for whom the therapeutic effect of ECT could be assessed. The median threshold for the entire group was a point somewhere between 3 and 3·5 mg/kg. Thirteen of the seventeen patients for whom a low threshold was

predicted fell below this point, whereas only one of the eleven patients for whom a high threshold was predicted fell below it. The concordance of test thresholds with prediction was highly significant statistically.

As regards improvement with ECT, the data in Table 2 show that the incidence of low thresholds was greatest in the "marked" and least in the "slight or no" improvement groups. A *t*-test for significance of the mean difference between the "sleep" thresholds of the "marked" and "slight or no" improvement groups, which was 1·63 mg/kg, almost achieved the 5 per cent level of significance. On the whole then, the present "sleep" threshold data conformed to the expectations based on previous studies with sedation and "sleep" thresholds.

"Sleep" threshold changes and clinical events

The organization of this section of results will reflect the form of analysis which was carried out. It was necessary first to look for relationships between clinical events and changes in the "sleep" threshold. Hypothesized relationships or "hunches" from the survey of individual cases were then subjected to statistical test. The results of five illustrative cases will be presented first.

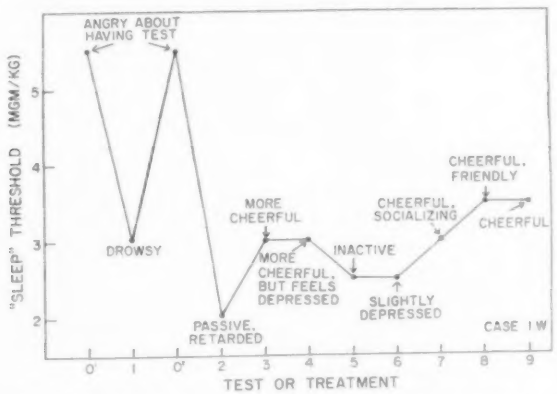

FIG. 3.—Case I.W. Note high thresholds in tests 0^1 and 0^2 in association with anger.

Individual case studies

Case I. W. (Fig. 3.) This 43 year old woman was admitted 12 March 1958, on referral from another hospital, where, after a 5 day stay, it was felt that she required ECT. The patient complained of: insomnia, anorexia, fatigue, headache, loss of interest, numerous crying spells, irritability and easy anger, amenorrhea for 3 months. She had a diurnal fluctuation, and expressed some suicidal ideas. She had become agitated and unable to work. She had also developed increased obesity, gaining over 20 lb in 6 months. It was learned that the body of her husband had been found in the river in September 1957, after he had been missing for 5 days. The patient believed that he had been killed. Two days after this her mother died from a stroke. The patient's symptoms began about 1 month after her mother's death. During her admission the patient expressed a great deal of hostility towards the hospital and the husband's family.

When approached for the pre-treatment test on 13 March, she expressed much anger about the test, stating that she "did not trust doctors in the hospital." She was actually prepared to have a treatment at the time of the test, as she had been informed that the treatment had been ordered; she considered the test unnecessary. "Sleep" threshold for this test was 5·5 mg/kg. About 4½ hr after the test, the patient was given her first ECT. At that time she seemed quite drowsy and her "sleep" threshold was 3 mg/kg. At the time of the treatment she expressed disgust about having so many needles, and

stated that she was somewhat afraid of her treatment. On the next day, a second test was done; the patient was again tense and hostile, although she did not express her hostility so openly, and once again, as in the first test, the threshold was 5·5 mg/kg. At no other time during the course of the patient's treatment was the "sleep" threshold higher than 3·5 mg/kg. The highest thresholds were for the last two treatments, both of which were 3·5 mg/kg. The patient showed quite a good response to treatment, apparently losing most of her symptoms.

Comment. The outstanding feature of this case was provided by the marked rises in threshold obtained in the non-treatment tests, about which the patient was very hostile. These suggest an association between anger and increase of the threshold. At no other times during the tests performed on this patient were similarly high thresholds obtained. It is not likely that the low threshold for the first treatment reflected some residual sedative effect of the test injection given earlier in the day, as later thresholds before treatments were even lower. There was a slight rise in threshold for the last two treatments, in association with remission of symptoms.

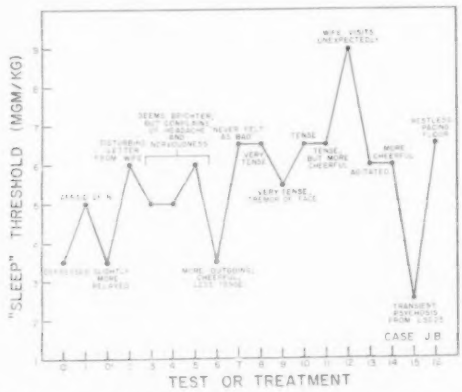

FIG. 4.—Case J. B. Note marked fluctuations in relation to fear of treatment (1), disturbing letter from wife and wife's visit (2, 12), apparent clinical improvement (6), and transient LSD psychosis (15).

Case J. B. (Fig. 4.) This 54 year old married man was admitted 18 January 1958, with symptoms of depression, anxiety and tension, which had been present in varying degree since 1956. He had received ECT in October 1956, at a hospital near his home in the Maritimes, and again 1 month later in a Montreal hospital. Treatment produced very little benefit. For 1 year the patient had been unable to work. He showed diurnal fluctuations, was anorexic, had lost considerable weight, was able to sleep only with the aid of pills. He had not expressed any suicidal ruminations, but was afraid of losing his mind. EEG was normal, sedation threshold was high (6 mg/kg). Psychological tests indicated depression associated with decompensating obsessional defences, and suggested that the patient was hostile toward his wife.

He was first placed on nitrous oxide inhalation therapy in an attempt to reduce his tension. With these treatments he wept bitterly, and said that he felt rather relieved. He began to speak about his wife saying that she had never been kind or understanding and had given him marked feelings of inferiority. He claimed that she had forced him to accept the idea that she was superior to him in every way. Because the amount of affect expressed with these statements was not very great, it was suggested that he be placed on Reiter convulsive non-convulsive therapy in addition to the nitrous oxide. (This was the only patient in our series who received treatment with the Reiter machine; his first nine treatments were of this type, the remaining seven being of the standard convulsive type.)

Prior to his first ECT the patient's ward behaviour was described mainly as consisting of agitation and depression, with pacing of the floor, poor appetite, marked restlessness and seeking of reassurance.

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This was the clinical picture on the day he received his pre-treatment test, although it was thought that there had been some improvement since admission. Throughout his course of treatment he expressed great fear of ECT, this fear having arisen during his previous treatments in 1956. The "sleep" threshold values for his non-treatment tests (0^1 and 0^2, Fig. 4) were 3·5 mg/kg, while the value for the first treatment test was 5 mg/kg. Between the first and second treatments, the patient received a very disturbing letter from his wife, which seemed to more than undo any improvement which he had shown up to that time. This letter reproached him in many ways, and the patient became more depressed and anxious. Another letter received after his second treatment was somewhat less disturbing and may have helped to produce a slight improvement in his general appearance from the third to the fifth treatment. However, although the patient seemed brighter to the nurses, he also complained a great deal, mainly of headaches and tension. Between the fifth and sixth treatments the patient seemed to be more outgoing and cheerful, and less tense, although there was still evidence of diurnal fluctuation. He participated more in ward activities. The threshold for the sixth ECT was low. Between the sixth and seventh treatments he complained of extreme tension, and stated; "I have never felt as bad." This state of manifest tension, with agitation, restlessness, poor appetite, overt tremor of the face and poor sleep, persisted over the next week, and was associated with increased thresholds. The patient seemed slightly more cheerful before the eleventh treatment, and it was noted that he had been telling some jokes on the ward.

However, this minor degree of improvement was very short-lived because on the day after the eleventh treatment his wife arrived unexpectedly. The interne spent nearly 2 hr talking with the wife, and felt that he could understand why the patient had so much difficulty with her. He described her as being taller and much heavier than the patient; she talked incessantly in a high, irritable, feelingless voice, smiling vacuously without humour. He thought her to be a stern, rigid uncompromising person, who had no regard for the feelings of others, and was herself completely devoid of insight. The "sleep" threshold for the twelfth treatment was the highest of any recorded for this patient, being 9 mg/kg. Clinically, during this time, the patient was very agitated. He continued to be agitated between the twelfth and thirteenth treatments, but was more cheerful between the thirteenth and fourteenth treatments. The "sleep" threshold for the fifteenth treatment was 2·5 mg/kg, lower than any other. The noteworthy event preceding this test was that, on the previous day, the patient had been given 100 μg of LSD 25, followed by a nitrous oxide treatment. He had returned to the ward with an unsteady gait, complaining that his eyes were out of focus, and expressing suspicions that people were laughing at him. He stated that the doctors had impregnated him with some germ. He was agitated, unable to sit or stand still and demanded to phone his wife immediately. He was given 50 mg of chlorpromazine intramuscularly, but continued to be suspicious, withdrawn, and expressing ideas of reference. Consequently, the fifteenth treatment was administered just after a transient psychosis. Before the final treatment the patient once again seemed agitated.

Comment. This case illustrates increases of the "sleep" threshold with fear, tension and hostility, and some tendency for decrease in the threshold with diminished tension and following a transient LSD psychosis. It may be noted that the patient had received LSD 25 on two other occasions, but that this was the only time that overt psychotic symptoms were produced by LSD, and also the only marked change in "sleep" threshold. It may also be noted that the patient was regarded as being distinctly anxious and apprehensive before every single one of his treatments, so that this factor was probably more or less constant for the entire period of observation. The lowered threshold after the LSD psychosis agrees well with the previously formulated concept that impaired ego functioning is associated with a low sedation threshold (SHAGASS, 1956).

Case B. B. (Fig. 5.) The parents of this 16 year old boy had observed a personality change about 2 months before admission. After failing in school he had become quiet, chewed his nails and paced the floor. He also complained of headaches. In the background it was noted that he had pulled out his eyebrows until he was seven and had had nocturnal enuresis until he was twelve. For 2 weeks prior to admission he had believed that people were laughing at him and thought that the shape of his head was wrong. There was frequent crying, but on the whole his affect was flat. Psychological

tests suggested an incipient schizophrenic process. His EEG was normal. Prior to the institution of ECT, about 1 month after his admission, the patient was treated with chlorpromazine. He was noted initially as being quiet and seclusive, but with medication he showed a good deal of improvement in his behaviour. However, when the medication was discontinued, he quickly relapsed, showing once again insomnia, restlessness, anxiety, fatigue, nail biting and ideas of reference. He seemed quite

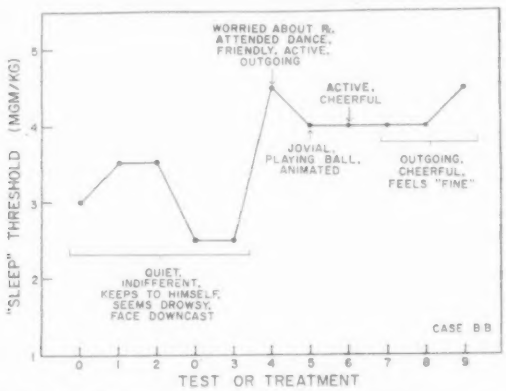
WORRIED ABOUT R, ATTENDED DANCE, FRIENDLY, ACTIVE, OUTGOING
ACTIVE, CHEERFUL
JOVIAL, PLAYING BALL, ANIMATED
OUTGOING, CHEERFUL, FEELS "FINE"
QUIET, INDIFFERENT, KEEPS TO HIMSELF, SEEMS DROWSY, FACE DOWNCAST
CASE B.B
"SLEEP" THRESHOLD (MGM/KG)
TEST OR TREATMENT

FIG. 5.—Case B. B. Note increased thresholds in association with clinical improvement.

indifferent to the test and also indifferent to treatment. With the first two tests and treatments he seemed somewhat drowsy, his face was downcast, he kept to himself and, when approached by the nurses, was regarded as seclusive. However, after the third treatment, two changes occurred. The patient's affect was no longer flat, and he expressed concern about the symptoms connected with his

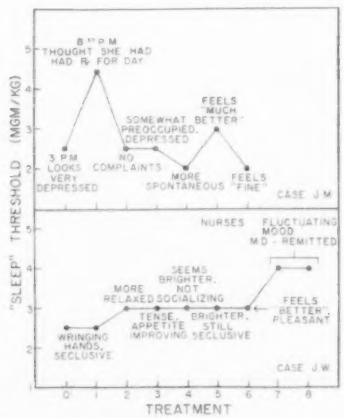

FIG. 6.—Upper part, case J. M. Note increase with reaction to unexpected treatment. Note also absence of further threshold changes, even though clinically improved. Lower part, case J. W. Note increase of threshold with improvement, and absence of marked fluctuations.

treatment. At the same time he attended a dance, seemed pleasant and friendly and joined in ward activities with interest. From this time on he was cheerful, outgoing, played ball and danced. He felt that he was quite well and wanted to go home. It will be noted that, whereas the "sleep" threshold for the third treatment was 2·5 mg/kg, the threshold for the fourth was 4·5 mg/kg and the thresholds remained elevated for the remainder of the series.

Comment. This case illustrates a rather close association between remission of psychotic symptoms and sudden elevation of the "sleep" threshold. Although the thresholds increased toward the end of treatment in most of the cases, and it was the general impression that there was an association between such an increase and clinical changes signalling the beginning of a remission, this is one of the few cases in which such an association was very clearly demonstrated.

Case J. M. (Fig. 6, upper half.) This woman of 60, had had symptoms of a deepening depression for about 6 months, with insomnia, diurnal fluctuation and retardation. Psychological tests confirmed the presence of a severe depression. Her EEG was found to be abnormal, containing a left temporal slow wave focus. Possibly associated with this finding, was the history of an old luetic infection. On the ward, prior to treatment, the patient was self-critical, pleasant, co-operative and rather passive; she complained only of constipation and kept to herself. Prior to her pre-treatment test she looked very depressed. Her first test was administered at 3 p.m. and her first treatment at 8.30 p.m. of the same day. When she was approached for this treatment she expressed surprise, saying she thought she had had her treatment for that day. She had apparently forgotten being told before the pre-treatment test that she would not receive her treatment till later on. The threshold for the pre-treatment test was 2·5 mg/kg, whereas for the first treatment it was 4·5 mg/kg. Over the five subsequent treatments she showed rather rapid improvement, feeling much better between the fourth and fifth treatments and apparently being fully remitted after the sixth treatment. As Fig. 6 shows, the only significant deviation in the "sleep" threshold was for the first treatment, at which time the patient was upset because she thought that she had already been treated earlier that day.

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Comment. The effect associated with the elevated threshold for the first treatment is not clear from the record, but one would wonder to what extent angry feelings were aroused. This patient is one of those who showed a clear remission with ECT, but who did not show any associated change in "sleep" threshold. In fact, there was a decrease from the first to last third of treatments, because of the high first treatment threshold. It is also noteworthy that, in contrast to I. W., whose threshold for the first treatment was considerably lower than the threshold for the test done on the same day, J. M.'s treatment threshold was higher. These differences seem more related to immediate affective reactions than to possible residuals of pentothal.

Case J. W. (Fig. 6, lower half.) This 59 year old man was admitted in November 1957, for treatment of this third depressive reaction, previous ones having been successfully treated with ECT in 1953 and 1955. He had been depressed for 3 months prior to admission, with complaints of anorexia constipation, weight loss, slowed thought processes, early waking. He had been drinking excessively in an attempt to control his symptoms. It was also felt that his depression had been precipitated by gambling losses. Psychological tests suggested a psychotic reaction with paranoid features. EEG was normal; sedation threshold was 3 mg/kg. Prior to commencement of ECT and the first "sleep" threshold he was tense, paced up and down wringing his hands and avoided others. "Sleep" thresholds for the test and first treatment were 2·5 mg/kg. Between the first and the second treatment he seemed to be more relaxed and then he gradually improved so that by the sixth treatment he admitted that he was feeling better and wanted to go home. Between the sixth and the eighth treatment the nurses recorded fluctuating mood, but his doctor felt that the patient's illness had remitted. The "sleep" threshold rose from 3 to 4 mg/kg between the sixth and the seventh treatment and stayed there for the eighth treatment.

Comment. The data on this case are presented to illustrate the hypothetical typical course of change in the "sleep" threshold with ECT. No unusual or complicating events occurred during the course of this patient's treatment, and he responded to treatment with a clinical remission which was associated with a terminal rise in threshold values.

Group statistics

The data for individual cases indicated that changes in the "sleep" threshold may frequently be associated with relevant clinical events. To assess the general applicability of this conclusion, the clinical events between all tests, as recorded mainly in nurses' ward notes, were coded independently by two of the investigators. Although

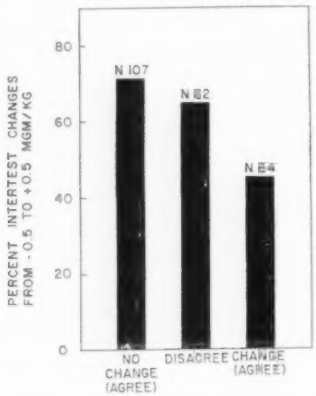

FIG. 7.—Percentage "sleep" threshold changes from −0·5 to +0·5 mg/kg between successive tests in relation to clinical assessments of change by two raters. Note that where raters agreed on change the incidence of minor threshold changes was lower.

"sleep" threshold data were not available to the investigators while coding, the possibility that there may have been some knowledge of these changes from previous contact with the data cannot be completely ruled out.

Agreement between raters. There were 273 inter-test intervals which could be coded. In 107 of these the raters agreed that no clinical change had taken place, and in 84 both agreed that some special event or clinical change had occurred. There was thus 70 per cent agreement on presence or absence of clinical change. Of the 84 agreements on change, 71 were coded under exactly the same categories; the coding differed in 13 instances, but in no case was a patient thought to be clinically improved by one rater and worse by the other. A good deal of disagreement occurred in connexion with the attempt to decide upon the beginning of a remission. One rater used this category 10 times, and the other only 3 times. However, in several instances where the remission category was used by one rater, the other coded clinical improvement.

Analysis of changes. The hypothesis to be tested may be formulated as follows: That periods associated with clinical change are associated with greater changes in the "sleep" threshold than periods in which no clinical change is observed. To support this hypothesis the variability of thresholds should be greater in periods of change than in periods of no change. As the probable technical error of the test is 0·5 mg/kg, individual deviations greater than 0·5 mg/kg may be regarded as significant. The prediction is that "change" intervals will contain more deviations between tests, which are greater than 0·5 mg/kg. Fig. 7 shows the proportions of deviations within the limit of error for those intervals when: (a) the raters agreed there was no change, (b) they disagreed about change, (c) they agreed there was change. It will be noted

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that the proportion of insignificant deviations for (b) was intermediate between (a) and (c), but more similar to (a). A chi square test showed the difference between (a) and (c) to be significant at the 0·1 per cent level of confidence. The difference between the change intervals (c) and the ones upon which there was disagreement (b) was significant at the 2 per cent level of confidence; (a) and (b) did not differ significantly. The *F*-ratios for difference between the variance of the three distributions were also calculated. The variance of the agreed change intervals was 2·66 times that for the agreed no change intervals and 2·75 times that for the intervals where there was disagreement. Both ratios were significant at the 1 per cent level of confidence.

The data thus supported the hypothesis of an association between clinical change and "sleep" threshold change. Of 106 "sleep" threshold changes of 1 mg/kg or more, 46 (43·5 per cent) occurred during the agreed change interval, 29 during intervals about which there was disagreement, and 31 during intervals when no change was agreed upon. Considering the almost accidental nature of many of the events counted as clinical change, and the obvious deficiencies in clinical reporting, it seems noteworthy that almost half of the significant "sleep" threshold deviations could be accounted for by clinical observation.

TABLE 3.—DISTRIBUTION OF "SLEEP" THRESHOLD CHANGES IN RELATION TO CLINICAL EVENTS AGREED ON BY TWO RATERS

"Sleep" threshold change (mg/kg)	No change	"Special" events	"Better", remission	"Worse"	Fear of treatment
−1·0 or more	13	8	6		3
−0·5	23		5	1	1
0	32		10	7	3
0·5	21		3	3	2
1·0 or more	18	7	6	5	4
Total	107	15	30	16	13

Examination of the coded data showed that the "agreed change" categories which contained most of the data could be grouped under four headings. These were: "special" events, "better" and remission, "worse", and fear of treatment. Condensed distributions of threshold deviations in these categories are shown in Table 3 together with the distribution for "no change". These data indicate that most of the difference in variance between the change and no change intervals was contributed by "special" events and fear of treatment. Comparison of the "better" and "worse" columns suggests a tendency for more threshold increases to occur in association with increase of symptomatology. The difference between "better" and "worse" categories was significant at the 5 per cent level, on the basis of a chi square test on a 2×3 table. It may be remembered, however, that clinical improvement at the end of treatment was generally associated with increased thresholds.

Following are the reasons for designating the "special" events items: hostile reactions to the tests ostensibly because they delayed treatment (case I. W.); disturbance because treatment was thought to have already occurred (case J. M.);

disturbance over wife's visit (case J. B.); LSD psychosis (case J. B.); reaction to losing a nephew before coming for treatment; disturbance because the doctor had difficulty with veins; reactions to week-end visits with a hated husband. Because the changes associated with these events did not seem to extend into subsequent periods, it was thought reasonable to include also the changes between the "special" events tests and the following tests in this change category. As "special" events sometimes occurred in the first test, the number of items was not exactly double the number of "special" events. It will be noted that most of these events involved a definite or probable hostility reaction and were associated with an increased threshold.

DISCUSSION

It is of interest to compare the present results with available data for the EEG-determined sedation threshold. As regards test–retest reliability, the average reliability coefficient for successive "sleep" thresholds was somewhat less, but probably not significantly so, than the coefficient of 0·83 obtained for thirty-seven patients who did not receive ECT, and who were tested on two occasions with the sedation threshold (SHAGASS, MIHALIK and JONES, 1957). Only a few patients have had numerous sedation threshold tests, and these also showed fluctuations of threshold in relation to changes in clinical status (SHAGASS *et al.*, 1957). In a study of the effect of ECT on the sedation threshold, forty-three patients received a total of 150 tests, these being done on days between treatments (SHAGASS, 1958). There was a statistically significant tendency for the sedation threshold to increase after ECT which is in agreement with present "sleep" threshold results. However, the present study did not confirm the finding that the increase in sedation threshold after ECT was a function of pre-treatment threshold, only patients with low thresholds (2·5 mg/kg or less) showing an increase.

It is not known whether the sedation threshold can change as rapidly or as much as the "sleep" threshold. It would nevertheless seem reasonable to recommend that both procedures should be carried out under relatively standardized environmental conditions. Another implication of the demonstrated potential variability of these determinations is that a single test, administered for diagnostic purposes, may reflect no more than a transient affective reaction. This possibility must always be considered, but it should *not* lead to a negative attitude concerning the application of sedative tolerance tests in psychiatric diagnosis. The relationships between these determinations and diagnosis have been consistently demonstrated in several sedation threshold studies involving hundreds of tests (SHAGASS and JONES, 1958; SHAGASS, 1958), in the study with the amobarbital "sleep" threshold (SHAGASS and KERENYI, 1958), and even in the present small group of patients. It may be well to emphasize that these relationships are *statistical*, and that there was always a considerable range of thresholds for any diagnostic category. This means that, under the conditions of testing, psychiatric disorders tend to be associated with sedative tolerances which vary within characteristic ranges. The measure of sedative tolerance itself apparently reflects dynamic, not static, processes, which may be relatively stable under some conditions, but may change radically under other conditions. The characteristics of stability and changeability may each serve useful investigative purposes, provided enough is known about their requisite conditions.

The main problem involved in devising a test of sedative tolerance is to find a

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suitable indicator of the threshold or end-point. At least four indicators of barbiturate action have so far been employed for this purpose: slurring of speech, the inflexion point in the amplitude curve of EEG fast-frequency activity, nystagmus to lateral gaze, and unresponsiveness to verbal stimulation. Slurring of speech, which was used as an auxiliary indicator in initial studies of the sedation threshold, has proved to be difficult to judge and seems to be of little value (THORPE and BARKER, 1957; FINK, 1958; SHAGASS and NAIMAN, 1956). The EEG sign of the sedation threshold is potentially the most objective indicator, as it involves a permanent record. However, aside from the difficulty of the method, which requires extensive facilities, one always obtains a certain proportion of equivocal records, even when the most careful attention is paid to details of measurement. The failure to obtain unequivocal determinations has occurred more frequently in some investigations than in others (FINK, 1958; PAMPIGLIONE, 1958; SHAGASS and JONES, 1958); it must be regarded as an undesirable limitation of the EEG method. FINK (1958) found that onset of lateral gaze nystagmus occurred within 0·5 mg/kg of the EEG inflexion point in 86 of 91 sedation threshold determinations. This observation is a further confirmation of the validity of the EEG inflexion point as an indicator of a certain level of central nervous system depression, and it suggests that nystagmus might be substituted for the EEG indicator of the sedation threshold. However, it seems likely that the clinical skill involved in judging nystagmus may not be equally present in all observers. The judgement of unresponsiveness used for the "sleep" threshold seems to require relatively little skill or training, and, providing enough drug is given, a threshold determination is always obtained. Other investigators have already incorporated similar "sleep" criteria in sedation threshold studies (BRADLEY and JEAVONS, 1957; PAMPIGLIONE, 1958). It would then seem that, of the indicators so far used, unresponsiveness may prove to be the most practical in terms of ease of application and actual repeatability of results.

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As a test drug, pentothal has a great advantage over amobarbital by virtue of its much briefer duration of action. The patient is thus incapacitated for only a short time, and more frequent testing is possible. However, although no untoward incidents occurred during the course of this study, pentothal is known to involve a greater risk of laryngospasm. The injection rate used here was probably rather rapid; it was selected as a compromise between the needs of the investigation and the treatment situation. A slower rate would probably allow a greater range of variation between tests and offer more possibilities for differentiation.

It should be obvious that a very large number of sedative threshold tests may be devised by using different combinations of drugs, rates of administration and indicators of end-point. Results so far suggest that most threshold tests using barbiturates should probably yield results of a similar nature. Application of the threshold principle to devise tests using other kinds of drugs acting on the central nervous system, such as excitants and ataractics, would seem to offer a promising field for psychiatric investigation. Progress along these lines will depend upon the development of useful indicators of threshold.

It is tempting to try to relate the finding that "sleep" and sedation thresholds increase with ECT to the phenomenon of "ECT anxiety," which ALEXANDER (1953) considers "the most important psychophysiologic complication" of ECT (p. 223), and which, according to GALLINEK (1956), occurs in a majority of patients. Although

it was hoped that the present study might provide data to bear directly on this problem, this hope was not fulfilled because clear definition of the time of emergence of "ECT anxiety" was not possible. Almost half the patients gave indications of fearing treatment, but this was nearly always noticed first right at the start, and generally persisted throughout therapy. Initial observations of fear were associated with increased variance of 'sleep' thresholds, but the thresholds decreased as well as increased. Indirect considerations do suggest that there was probably no consistent close temporal association between the development of "ECT anxiety" and increase of the "sleep" threshold. Although the mean threshold increased progressively, the first statistically significant increase over the initial treatment threshold was found at the seventh treatment. This would be considerably later than the expected exacerbation of anxiety at about the third or fourth treatment (ALEXANDER, 1953). However, even if the two phenomena are not directly related in time, it would still seem possible that both represent manifestations of increased cerebral excitability brought about by ECT.

The hope that changes in the "sleep" threshold might be of value as an indicator of favourable or unfavourable therapeutic progress with ECT was also not supported by present data. Some patients improved in close association with increased "sleep" thresholds, but this was not a consistent finding.

Anger or hostility seemed to be associated with the most striking increases of "sleep" threshold observed in this study. Such increases could apparently take place in, at most, a matter of minutes. This association between raised "sleep" threshold and hostility seems to coincide with the commonplace experience that it is hard to fall asleep when one is angry. It also suggests a possible explanation for the consistent finding that patients with acute psychoses have low sedation thresholds (SHAGASS, 1956; SHAGASS and JONES, 1958), even though this is difficult to reconcile with the fact that enormous amounts of sedation may frequently be required to quieten a very excited patient. It seems likely that the failure to test patients who are too excited to co-operate may account for the absence of high thresholds in this group. Before doing "sleep" threshold studies, it was thought that in such cases there might be a dissociation between sedation and "sleep" thresholds, the former being low and the latter high. However, the "sleep" thresholds of acute psychotic patients were generally found to be low in initial testing. They increased in association with anger and such increases could double the threshold even though the degree of anger observed in our patients was not nearly as great as the extreme hostility shown by many excited psychotics.

Present data failed to confirm the prediction that "sleep" thresholds at the time of the first ECT would be higher than those obtained in preceding and following tests not associated with ECT. This seems to have been due mainly to the fact that a variety of unpredicted emotional reactions occurred in what seemed to be a relatively predictable situation. Some patients seemed more disturbed by the "control" tests than by their first treatment, some seemed to react more to an event like the doctor having trouble finding veins, others showed little or no emotional reaction to either test or treatment. Even though the emotional effects of the ECT situation appear on the surface less unpredictable than those of an interview situation, present experience seems to coincide with that of GRINKER's group in their attempt to manipulate anxiety experimentally (GRINKER *et al.*, 1957). One must conclude that the experimentalist's

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ideal of planned induction of emotional reactions is most difficult to achieve. The findings of this study which do show a relationship between affective reactions and "sleep" threshold changes were obtained by virtue of frequent tests on reactive subjects under conditions conducive to affective change. There was thus a high probability of occurrence of "experiments of nature," which would be relevant to the purpose of the study.

SUMMARY

(1) In this investigation factors influencing variation of a simple modification of the sedation threshold, designated here as the pentothal "sleep" threshold, were studied in relation to the situation of electroconvulsive therapy. The aims were: (a) to assess the stability of the "sleep" threshold; (b) to determine the effect of ECT on the threshold; (c) to determine whether the threshold reflected affective changes.

(2) The "sleep" threshold was taken as the amount of intravenous pentothal, injected at the rate of 0·5 mg/kg per 30 sec, required to produce unresponsiveness to verbal stimulation. Twenty-eight psychiatric patients received 306 tests, of which 259 were used as premedication for ECT. Eleven additional tests were carried out in the presence of from three to five observers in order to assess difficulty in judging unresponsiveness.

(3) Results were as follows: (a) In most instances, observers deviated by no more than 0·5 mg/kg in judging unresponsiveness. (b) Correlation coefficients between "sleep" thresholds obtained in successive tests ranged from 0·57 to 0·90, and averaged about 0·75. (c) Thresholds increased significantly from start to end of ECT. This increase did not occur in all patients, was not a function of pre-treatment threshold values, and was not correlated with therapeutic effectiveness of ECT; it was somewhat correlated with numbers of treatments. (d) Pre-treatment pentothal "sleep" thresholds agreed with psychiatric diagnosis and outcome with ECT in the directions predicted from previous studies of the sedation and amobarbital "sleep" thresholds. (e) Contrary to prediction, the "sleep" threshold for the first ECT was not significantly higher than thresholds determined in preceding or succeeding non-treatment tests. (f) Reactions of hostility and anger were associated with increased thresholds. There was also a tendency for a relative increase in thresholds with intensification of symptoms, when compared with changes associated with improvement of symptoms and behaviour. On the other hand, clinical remission was at times closely associated with increased thresholds. (g) Affective changes and reactions, noted clinically, were significantly associated with "sleep" threshold changes. This was observed both in study of individual cases and for the group as a whole.

(4) It was concluded that: (a) The pentothal "sleep" threshold is a reasonably reliable determination, which may give diagnostic and prognostic results simliar to those obtained with the sedation threshold. (b) From the point of view of ease of application, the "sleep" threshold seems to be the most useful measure of sedative tolerance so far investigated. (c) ECT increases the "sleep" threshold, but the ongoing changes in the threshold cannot be used as a guide to favourable or unfavourable progress of treatment. The increase probably reflects a non-specific central effect of ECT in increasing brain excitability. (d) The pentothal "sleep" threshold is very sensitive to certain affective changes, notably those connected with hostility, and may serve as a useful indicator of such reactions.

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AN EVALUATION OF THE SEDATION THRESHOLD TEST

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EMOTIONAL states when investigated experimentally are best treated as subjective epiphenomena which may occur concomitantly with bodily changes, both being accompaniments of stressful adaptations. We cannot, however, quantify the subjective state, and the behavioural expression of emotion is affected by cultural factors and influenced by the immediate environmental situation. We do not even know to what extent the various bodily changes are intercorrelated and it is doubtful whether changes in one field can be reliably accepted as an indication of changes in others. Nor can we quantify "emotional stress" which is dependent on factors both without and within the individual. We can expose subjects to comparable stimuli or environmental factors, but it is the significance of the latter to the individual which determines whether at any particular time they will be stressful or not.

Rather than attempting studies on induced stress, the investigation of changes in certain physiological variables in emotionally disturbed subjects under conditions of sedation has appeared to some workers to be a more profitable line of enquiry. This approach is the basis of the test devised by SHAGASS (1954) and named by him the "sedation threshold". Starting from the assumption that the more tense was a subject the greater would be the quantity of sedation required to produce a relaxing effect, he deduced that it would follow that the amount of sedative required to attain a specified end-point of sedation could be taken as a quantitative index of degree of tension present. In order to define an objective end-point of sedation the clinical assessment of the onset of slurred speech was combined with the observation of certain changes appearing in the EEG frontal fast activity, both phenomena being induced by intravenously injected barbiturate.

The technique developed by SHAGASS involved the intravenous injection of Sodium Amytal at a rate of 0·5 mg/kg body-weight every 40 sec, the subject being asked 25 sec after each injection to repeat "5–6–7", "77", "Massachusetts". The injection was continued until the onset of slurring of speech and interrupted after about two further injection intervals. The sedation threshold was defined as the amount of Sodium Amytal in mg/kg body-weight required to produce an inflection point in the amplitude curve of frontal 15–30 c/s activity; the inflection point coincided with the clinical observation of slurred speech in 76 per cent of cases, while in the remainder it did not differ by more than 80 sec. The inflection point was defined as that point which followed an abrupt rise and preceded a tendency to flatten, or the point just preceding a clear plateau in the curve. It was considered that the clinical observation of slur gave an approximate identification of the threshold which was more accurately defined by the EEG change.

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In a series of publications SHAGASS has expanded the application of the sedation threshold test. The manner in which he has successively developed the interpretation of his findings will now briefly be described.

The sedation threshold was first found to be correlated with "tension" as judged clinically, using neurotic and control subjects. Subsequently the term "manifest anxiety" replaced "tension" and it was considered that it should be greatest in anxiety states and neurotic depressions, least in conversion hysterics, with the mixed neuroses intermediate. This expectation was found to conform to the sedation threshold results for different neurotic groups (SHAGASS and NAIMAN, 1956).

Hysterical personalities, in common with patients having hysterical conversion symptoms, tended to have a low threshold. However, obsessional personalities were found to predominate in anxiety states and the latter have high thresholds. SHAGASS therefore considered that the sedation threshold was correlated not only with the degree of manifest anxiety but also with a hysteric–obsessional personality continuum. He suggested that his test "may reflect some basic neurophysiological aspect of personality organization" and added that "not only the order but the direction of the sedation threshold differences agrees with Eysenck's data and theory on introversion–extraversion" (SHAGASS 1956; SHAGASS and KERENYI, 1958).

The threshold was not found to be significantly correlated with degree of tension in schizophrenics, but the recent acute cases were found to have lower thresholds than equally tense chronic ones. Considering that in "acute psychoses, ego functions are thought to be more impaired than in non-deteriorated psychoses of long duration, in which reparative processes have taken place" the hypothesis was formulated that the sedation threshold was negatively correlated with degree of impairment of ego functions. According to this hypothesis, the greater the degree of ego impairment, the lower the threshold (SHAGASS and NAIMAN, 1955). The sedation threshold in various psychotic categories was again found to conform to expectation, the organic cases having the lowest thresholds, followed by the acute schizophrenics, the agitated depressives and the chronic schizophrenics, with the "borderline states" having the highest threshold (SHAGASS, 1956).

This concept of impairment of ego function was used to explain why agitated depressives (also called psychotic depressions), who are very tense, have low thresholds, whereas "neurotic depressions, providing they do not occur in severe hysterics, should have high ones". The sedation threshold is claimed to distinguish psychotic depressions from neurotic depressions and anxiety states, and to be able to give a good indication of the "immediate response" to ECT (SHAGASS, NAIMAN and MIHALIK, 1956).

However, no correlation could be demonstrated between sedation threshold test results and those from FUNKENSTEIN *et al.* (1952) test of autonomic function which is also claimed to predict prognosis with ECT (SHAGASS and JONES, 1958).

SHAGASS *et al.* (1957) reported that, whereas the sedation threshold was thought to be very stable on repeat testing, it is in fact found to decrease in some subjects where the degree of affective disturbance diminishes. Accepting that this must limit its value in the field of diagnosis and personality he states that the sedation threshold "may now be proposed as an objective index of therapeutic change". SHAGASS accepts that it is "difficult to arrive at a completely satisfactory formulation of the psychological factor which is positively correlated with the sedation threshold" and in an attempt to resolve various contradictions he has proposed the following assumptions:

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(1) That there are several neurophysiological mechanisms which may mediate reactions clinically grouped together as "anxiety"; of these the sedation threshold reflects only one.

(2) That personality structure plays an important role in determining which of these several mechanisms will mediate the anxiety reactions of a particular individual.

Granted these assumptions it may be stated that the sedation threshold measures the degree of activity in that mechanism of anxiety which predominates in obsessional individuals.

This statement suggests that only in such personalities is the sedation threshold a valid measure of anxiety.

In a later paper (SHAGASS and JONES, 1958), emphasizing that the sedation threshold must be applied within the context of other clinical information, SHAGASS states: "Like most laboratory procedures, it is influenced by and related to a variety of factors and does not mean much as an isolated datum".

In view of the claims made for the clinical use of the sedation threshold and the amount of attention it has attracted amongst pyschiatrists, it was decided to attempt an investigation of its value. Furthermore, one of us had undertaken a plethysmographic investigation which also used, though in a different manner, barbiturate sedation in the study of anxiety (ACKNER, 1956). It seemed, therefore, profitable to see whether the results of this study could be correlated with those of the sedation threshold and these findings have already been reported (ACKNER, 1958; PAMPIGLIONE, 1958). The present paper considers the sedation threshold test with particular reference to those methodological aspects which have a bearing on the evaluation of results.

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EXPERIMENTAL PROCEDURE

Certain modifications of the procedure, as described by SHAGASS, were introduced by us for the purpose of greater reliability and objectivity. A preliminary EEG (without barbiturate injection) was carried out a few days prior to the actual test to familiarize the subjects with the test procedure and reduce test anxiety. In order to minimize the disturbance due to the actual injection, a Gordh's needle was used. This is an intravenous needle with a rubber diaphragm at one end, which after insertion into the vein can be left strapped to the arm. By this means, it is possible to wait until the patient is suitably settled and then the injection is begun, without discomfort to the patient, by means of another needle inserted through the rubber diaphragm.

The verbal responses were registered on a tape recorder and the intravenous barbiturate injection was not interrupted by any impression of slurring gained during the course of the test. The injection was instead continued until no further verbal responses were obtained.

The subjects studied were all recently admitted psychiatric in-patients of the Maudsley Hospital. No subject was included if ECT had been administered in the preceding 6 months, if tranquilizers were being administered, if the superficial limb veins were unsatisfactory for injection or if marked restlessness was likely to interfere with the test procedure. Previous nocturnal sedation was not considered to be a contra-indication, but no sedatives were administered on the day of the test. The test procedure was satisfactorily completed in sixty patients, the average age for the whole group being 35 years.

The test procedure was carried out in one room with the majority of the recording apparatus housed in an adjoining room. The EEG was recorded on three channels, the amplification being a 5 mm pen deflection for 50 μV, with a time constant of 0·3

sec and a high-frequency cut of 15 per cent at 75 c/s. The electrode distribution was similar in all patients and was comparable to that used by SHAGASS (1954) who obtained his measurements recording from F3 to F4 (bifrontal) of JASPER's "10–20" system (1941) or from sagittal fronto-central electrodes.

In addition records of the finger plethysmogram and the respiration were taken at the same time as the EEG.

After the EEG electrodes had been applied, the patient was allowed to relax for a period and then was interviewed by one of us (B. A.), and by another psychiatrist assisting with the injection procedure. As judged by his appearance, behaviour and statements about his feelings at the time the patient was rated for "manifest anxiety" on a 0–5 scale.

Subsequently the Gordh's needle was inserted and after a suitable time to allow the patient to settle. Sodium Amytal was injected at a rate of 1 ml/40 sec, each millilitre containing 0·5 mg/kg body-weight.

One of us (G. P.) was in the adjoining room supervising the recording and being able through earphones to monitor the injection proceedings. All the records were marked during the test procedure for the time of the insertion of the needle, the first millilitre injected, each further millilitre, the verbal responses, the time of unresponsiveness, the end of the Sodium Amytal injection, the time of injection of Megimide (and any further such injections at 3 min intervals), any stimuli applied, and the time of awakening. At regular intervals the figure on the dial of the tape recorder was entered on the EEG record, thus enabling subsequent correlation. Twenty-five seconds after each injection, the patient was asked to repeat "57", "5–6–7", "British Constitution" (the latter was considered to be more suitable for our subjects than Massachusetts!). When the patient finally ceased to respond, one further injection was given and after a short interval consciousness was restored by means of an intravenous injection of Megimide.

Immediately afterwards a determination was made of the time at which the patient had slurred, this being defined as the first time a definite disturbance of articulation could be noted. Starting at various points, the tape recording was played back on a number of occasions until the decision was jointly agreed by the three doctors and the recordist present. The figure on the dial of the tape recorder was then read and the corresponding figure on the continuous paper recording was found. By this means the amount of Sodium Amytal injected at the time of slurring could be derived, the slur point having been determined uninfluenced by knowledge of the course of the injection.

EEG measurements were carried out with the aid of the special ruler designed by SHAGASS (1942) to count waves and add up their amplitude. This ruler was found to be simple to use but measurements on one record could take up to 2 hr to carry out. The growth curve of the barbiturate-induced frontal fast activity was obtained by measuring certain samples in each 40 sec injection period and plotting the mean amplitude against the amount of Sodium Amytal in mg/kg body-weight injected. Following the method of SHAGASS, the amplitude of all waves from 15–30 c/s was measured in samples of consecutive 2 sec of tracing which contained the maximum fast activity in each 40 sec injection period. A decision as to which sample contained the maximum fast activity involved the possibility of subjective bias, and in order to avoid this danger measurements were also carried at a fixed interval, namely, in the

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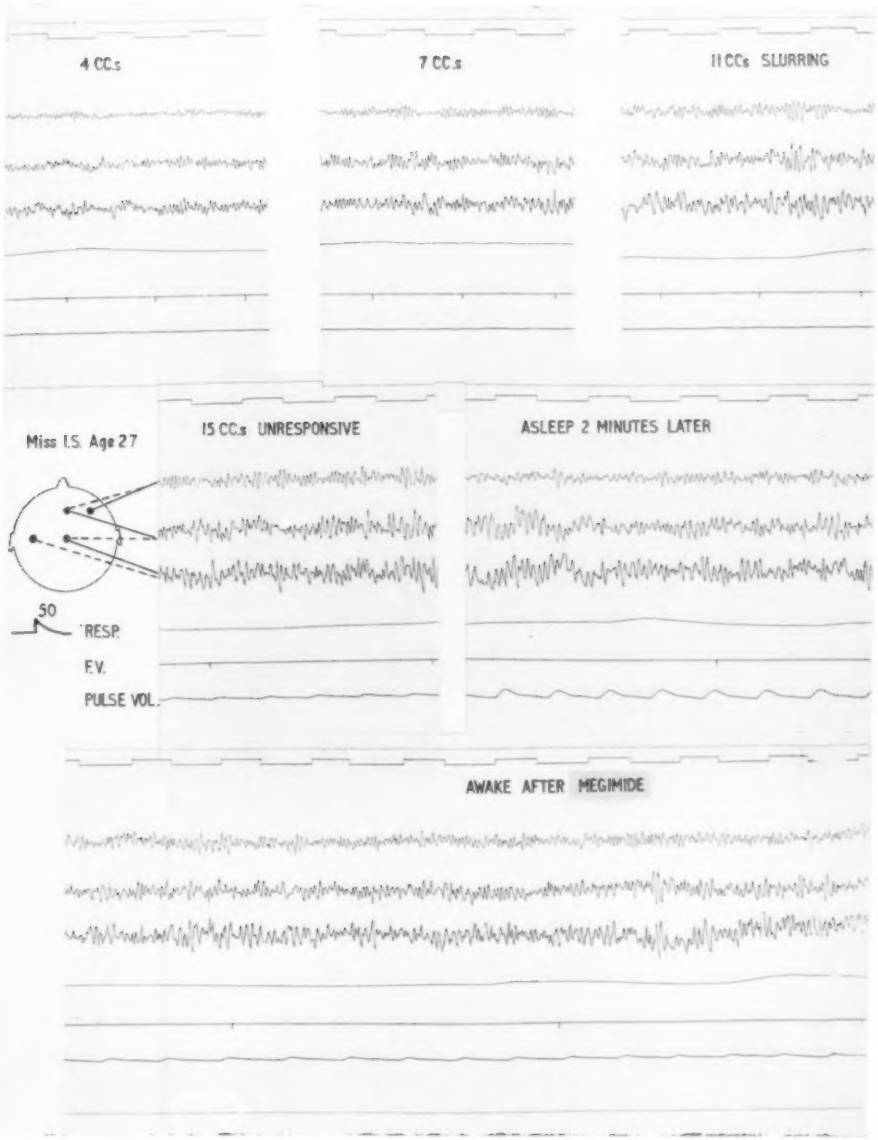

FIG. 1—Early appearance of fast activity in all EEG channels with considerable increase in amplitude. This record also demonstrates the increase in finger pulse volume occurring during early sleep.

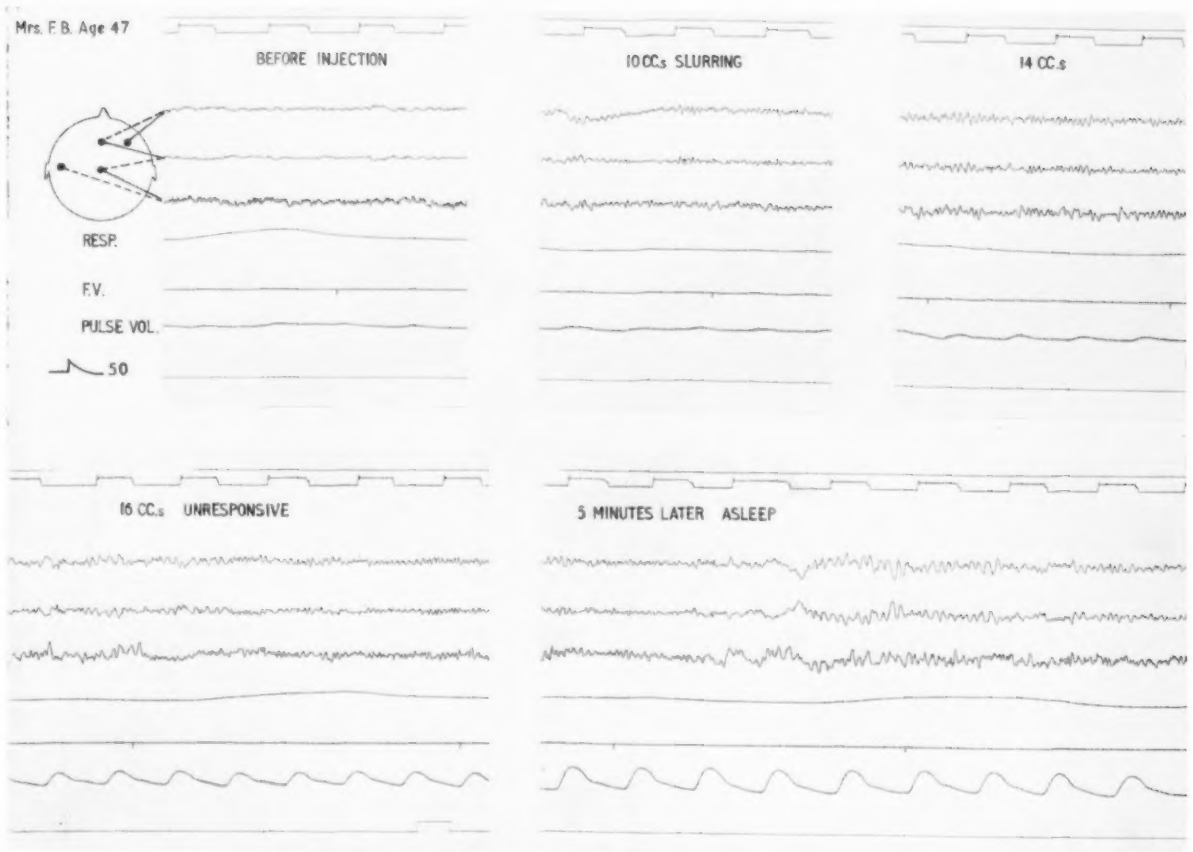

FIG. 2.—Late appearance of fast activity in all EEG channels with small increase in amplitude. This record also demonstrates gradual increase in finger pulse volume during injection.

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2 sec preceding each millilitre injected. The growth curves derived from these two methods of sampling were similar and the variation did not exceed 20 per cent. The range of error on repeat measurement of the same samples was found also not to exceed 20 per cent and this is of the same order as that found with wave analysers.

RESULTS

Appearance and increase of fast activity in the EEG

During the injection there was in all patients an increase in amplitude of 15–30 c/s activity considered to be a specific response to barbiturates. There was, however, considerable variability between patients in the time of appearance of such increase of fast activity. In some the fast activity was increasing as early as ½ min after the second millilitre injected (Fig. 1), while in others no change occurred before 8–9 ml had been injected (Fig. 2). In more than half of the cases (36 out of 60) the increase in amplitude of fast activity was seen after 4–5 ml (2–2·5 mg of Sodium Amytal/kg body-weight).

Once the fast activity had appeared, or began to increase, there were considerable fluctuations in its amplitude over the 40 sec interval between each millilitre of solution injected. These fluctuations in amplitude did not appear to be related to any obvious factor such as the patient's expectation of the test words or his replies.

It was, however, noticed that the growth curve of the fast activity recorded from the right frontal electrode to the midline was not always similar to the growth curve of the fast activity recorded from the midfrontal electrode to the vertex. In 6 patients records were taken both from the right frontal to the midline electrode and from the right to the left frontal electrode. Some difference in amplitude was seen between these two channels, the amplitude being slightly greater recording from the right and left frontal electrode than recording from the right frontal to the midline electrode. This was attributable partly to differences in inter-electrode distances and partly to less clearly understood factors such as the spatial and phase distribution of fast activity in various regions. However, no marked differences were seen between the shape of the growth curves in either channel.

The increase in fast activity measured from the same channel in each patient was not proportional to the amount of drug injected, and in some the rise was steep while in others the increase was very slow (Figs. 1 and 2). In some the rise was of short duration while in others it was of long duration. Fluctuations in the rate of increase of fast activity were common and only in 22 patients of the 60 did the curve have some resemblance to the S-shaped pharmacological curve described by SHAGASS (Fig. 3). In most of the others the growth curve had an irregular outline with several inflection points (several periods of increase and decrease of the mean amplitude of the fast activity). In 5 patients there was only a minimal increase in fast activity with an almost flat curve throughout (see Fig. 4). By the time the patient became unresponsive the fast activity had reached in most cases its maximum amplitude and there was usually a tendency to a gradual diminution thereafter.

Determination of slur

In spite of our precaution of using a tape recorder and of defining slur as the time of the first disturbance of articulation, the assessment of slur proved to be difficult.

It was impossible in 11 patients, 4 of whom did not show disturbance of articulation before suddenly becoming unresponsive, 3 abreacted, and 4 exhibited an indistinct speech at the time of the test, without any definite deterioration. Of the remaining 49 patients 27 slurred on one occasion but appeared to improve soon after, showing a further gross slur after one or more additional injections. In the remaining 22, slurring became gradually worse after its first appearance. In some patients the speech altered in tone or became slower, more deliberate or more hesitant, and to distinguish these changes from disturbances of articulation was sometimes difficult. Furthermore, it was apparent that, without careful control, the examiner's mode of verbalization (e.g. firmness and loudness of speech) could affect the nature of the

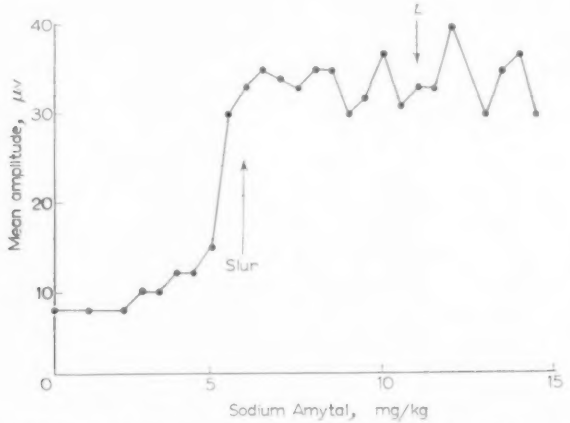

FIG. 3.—Growth curve of EEG frontal fast activity similar to that described by SHAGASS. The inflection point occurs close to the time of slurring. (L = last injection of Sodium Amytal.)

subject's response. On many occasions the record on the tape had to be run four or five times before a joint decision could be agreed on the time of slurring. There was no definite way of predicting, once the patient had slurred, whether his speech would become better or worse after the next injection, or whether he might fail to respond any longer. Nine patients failed to respond on one occasion but began to respond again after the next millilitre had been injected. The injection was interrupted when the patient failed to respond on two consecutive occasions.

Determination of the sedation threshold

The time of the first disturbance of articulation was compared with the time of the "inflection point" in the growth curve of the fast activity in the EEG. According to SHAGASS the two should coincide in the majority of cases and in all cases slurring should occur within the injection of 1 mg/kg body-weight (80 sec) from the inflection point. Of the 60 patients, 11 could not be used because of the lack of a definable change in their speech during the test. In the remaining 49 patients the EEG growth curve failed to show any inflection point within 80 sec of the slur in 16 cases. There were therefore only 33 patients out of 60 in whom the sedation threshold could be examined and of these 9 at least showed a growth curve with more than one inflection point near the time of slur, thus making difficult any precise determination.

Determination of the time of unresponsiveness

This was assessed, as mentioned above, as the time at which the patient failed to respond on two consecutive occasions. There was a considerable variability between patients and some fell asleep as early as after the injection of 12 ml (6 mg/kg body-weight) while others required 30 ml or more of the solution matched to their body-

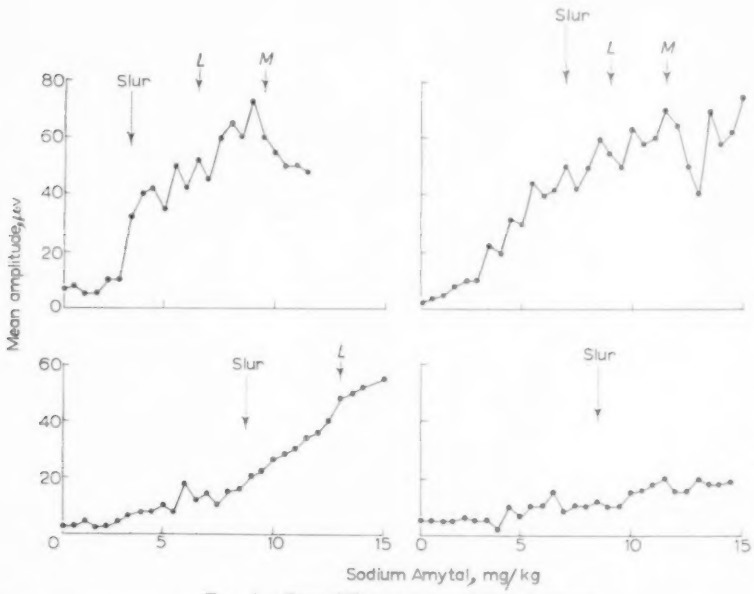

FIG. 4.—Four different types of growth curve:
(a) Steep rise and inflection point close to the time of slurring.
(b) Gradual rise with several inflection points. N.B. In this type of curve, wherever slur might occur it would always be closely associated with one of the inflection points.
(c) Gradual rise with slur unassociated with clear inflection point.
(d) Minimal rise with slur unassociated with clear inflection point.
(*L* = last injection of Sodium Amytal); (*M* = injection of Megimide)

weight. In the whole group all but 3 patients fell asleep with less than a total of 1 g of Sodium Amytal, with an average of 19·6 ml (9·8 mg/kg body-weight). There was a considerable similarity in the average amount of drug required both for patients with a rather low body-weight and for those with a rather high body-weight, in spite of the marked scatter within each group.

Anxiety rating (*see Table* 1)

It was found that the 0–5 rating scale for anxiety produced too many small groups and so this six-point scale was reduced by combining ratings 0–1, 2–3, 4–5, to give three groups which represented clinically minimal (0), moderate (+) and marked anxiety (++). The sedation thresholds were widely scattered in each group but the mean sedation thresholds were 5·8, 5·3 and 4·8 respectively. These figures did not reveal any marked differences between the sedation thresholds of the three anxiety groups, though the trend was the opposite of that found by SHAGASS.

TABLE 1

Anxiety rating	0	+	+ +
Mean sedation threshold in mg/kg body weight (33)*	5·8 (10)	5·3 (16)	4·8 (7)
Mean 'unresponsive point' in mg/kg body weight (57)	9·5 (17)	9·5 (24)	10·2 (16)

* The figures in brackets (in this and following tables) indicate the number of cases in each category. The sedation threshold could only be determined in 33 out of 60 cases. In 3 out of 60 cases unresponsiveness did not occur with 1 g of Sodium Amytal and the injection was abandoned.

The amount of Sodium Amytal required to produce unresponsiveness was again widely scattered in each group but the mean amount for each group was again very similar being 9·5, 9·5 and 10·2 mg/kg body-weight respectively.

Diagnostic categories (*see Tables* 2 *and* 3)

The diagnostic grouping of the patients was carried out independently of any knowledge of the sedation threshold results, the criteria for the various categories being those described by SHAGASS. The categorization was not usually carried out until the discharge of the patient, when a fuller knowledge of the history and evolution of the clinical picture was available. Sedation threshold results were available for the following diagnostic groups:

(1) Anxiety states and neurotic depressions.
(2) Obsessional states.
(3) Mixed neuroses.
(4) Endogenous depressions.
(5) Hysteria.

As the sedation threshold could not be determined in nearly half our cases, for the reasons described above, the number of cases remaining in each diagnostic category was necessarily small. However, although there was a wide scatter in the

TABLE 2

Diagnosis	Mean sedation threshold (mg/kg body-weight)	Mean "unresponsive point" (mg/kg body-weight)
Anxiety state / Neurotic depression (8)	5·3	8·6
Obsessional state (3)	5·7	12·5 (2)*
Mixed neurosis (5)	5·1	8·3
Endogenous depression (8)	4·9	8·7
Hysteria (5)	6·1	10·4
Others (4)	5·4	8·1

* One failed to sleep.

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individual figures the mean sedation thresholds were similar in all groups. The hysterical group tended to have a higher mean threshold than the others and this is the reverse of what would be expected from the findings of SHAGASS. The mean amount of Sodium Amytal required to reach the point of unresponsiveness also did not show any marked differences between the groups.

TABLE 3

Diagnosis	Mean "unresponsive point" (mg/kg body-weight)
Anxiety state / Neurotic depression (19)	10·3
Obsessional state (4)	11·4
Mixed neurosis (8)	8·1
Endogenous depression (14)	9·0
Hysteria (6)	10·5
Others (6)	9·2

DISCUSSION

Certain difficulties in the estimation of the sedation threshold have emerged, and furthermore we have been unable to obtain the correlations with the anxiety ratings and diagnostic groups described by SHAGASS. The differences in our findings may perhaps be related to differences in experimental procedure.

The possibility that the assessment of the slurring point might be unconsciously influenced by the clinical impression made by the patient was eliminated by registering the verbal responses on a tape recorder. The injection was continued to the point of unresponsiveness and the occurrence of slurring was subsequently assessed. This method revealed that slur was unreliable as an end-point of sedation. THORPE and BARKER (1957) carried out a study in which they played back tape recordings of the speech of 7 patients during intravenous injection of Sodium Amytal according to SHAGASS's method. No agreement was found on the occurrence of slurring amongst 16 independent medical assessors.

In our cases the mean amount of Sodium Amytal required to produce slurring was much higher than that found by SHAGASS. This may be due to the more objective method used and to the criterion of slur in our cases being defined as the first definite sign of disturbance of articulation. However, LAVERTY (1958), also working with British subjects, found higher figures for slurring.

If then the assessment of slur is so unreliable it might seem reasonable to eliminate it altogether, for SHAGASS has claimed that it only gave an approximate identification of the threshold which was more accurately defined by the EEG change. This could be justified if, in fact, the growth curve of the EEG frontal fast activity was always of the S-shaped form, described by SHAGASS, having one clear inflection point. In practice, when the injection is continued until the patient ceases to respond, the growth curve is often found to be irregular in shape with a number of inflection points. If no slur point is available there is no means of deciding which of a number of inflection points is to be taken as representing the sedation threshold. This being

so, the estimation of the slur must be regarded as an integral part of the sedation threshold estimation and as such cannot be dispensed with.

It seems possible that the use of an analyser, sampling over a longer time interval, would have a "smoothing" effect on the growth curve, though it would not be likely to alter its general shape. However, in 500 out of 750 cases reported by SHAGASS and JONES (1958) the hand measurement method had been used and this method was reported as "accurate but rather laborious".

The assessment and rating of anxiety itself provides a number of methodological difficulties. SHAGASS originally used the term "tension" though later changed this to "manifest anxiety". However, it appears that in the assessment of the latter, not only were behavioural changes observed but, in addition, subjective complaints and various features of the clinical history were also taken into account. Under these conditions "anxiety" must be regarded as a concept, rather than as an operational term subject to quantification. In our attempts to rate anxiety we tried to restrict our criteria to the signs of anxiety actually manifested by the patient prior to the experimental procedure and we attempted to reduce the influence of "test anxiety" by previously exposing the patient to part of the test situation. However, we recognized how unsatisfactory are such rating procedures and when differences of opinion occurred it was necessary to average the rating assessments.

It may be that differences in rating procedure account for our failure to obtain correlations between anxiety and the sedation threshold. On the other hand the diagnostic groupings represent broad and fairly easily definable categories. There is, for example, usually little difficulty in deciding whether a case should be defined as an anxiety state, endogenous depression or conversion hysteria. Yet no clear correlations could be found between these diagnoses and the sedation threshold.

The assumption that the amount of sedative required to attain a specified end-point of sedation can be taken as a quantitative index of anxiety, is central to the concept of the sedation threshold. Another possible end-point of sedation could be taken as that stage of disturbance of consciousness at which a subject fails to give a verbal response. However, in our studies, the mean amount of Sodium Amytal (in mg/kg body-weight) required to reach the point of unresponsiveness was similar both for the anxiety groups and for the various diagnostic categories. It may be, therefore, that one of the basic assumptions on which the sedation threshold test rests is itself questionable.

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SUMMARY

The sedation threshold test devised by SHAGASS (1954) is based on the assumption that the amount of sedative required to attain a specified end-point of sedation can be taken as a quantitative index of tension. As an objective end-point of sedation the test employs the clinical assessment of the onset of slurred speech combined with the observation of certain changes appearing in the EEG frontal fast activity, both phenomena being induced by intravenous injection of Sodium Amytal.

The sedation threshold has been claimed to give an index of anxiety and of therapeutic change, to distinguish anxiety states and neurotic depressions from hysterical conditions, to distinguish between hysterical and obsessional personalities, to distinguish between neurotic and psychotic depressions and to indicate the immediate outcome of ECT.

In the present investigation the methodology was modified in two main respects.

Slurring of speech was assessed retrospectively by means of a tape recorder and was uninfluenced by any clinical impression of slurring made by the patient at the time of testing. The intravenous injection was not interrupted at the time of slurring but was instead continued until no further verbal responses were obtained.

This method revealed problems in the assessment of slurring and more variable EEG changes than those previously described. The combination of these difficulties made it impossible to determine the sedation threshold in more than 33 out of 60 cases. The results for these cases did not reveal any significant correlations either with the anxiety ratings or with the diagnostic groupings.

In 57 out of 60 cases it was possible to determine the amount of Sodium Amytal required to produce unresponsiveness. The results for these cases again did not reveal any significant correlations either with the anxiety ratings or with the diagnostic groupings.

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THE MECHANISM OF THE INCREASE IN DIURESIS INDUCED BY HYPNOTIC SUGGESTIONS*

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THE typical characteristic of the human psyche, of the higher nervous activity, is the second *signalling* system, the effect of words. This furnishes the basis for the methods of verbal hypnosis and suggestion. Although little is known of their mechanism, the changes induced by these methods in the function of internal organs can be measured precisely and reflect the influence on the function of internal organs of the activity of the most highly differentiated components of the human cerebral cortex, of the higher nervous activity in man.

MARX (1926) observed in patients with hysteria an increase in diuresis and in the dilution of blood on the hypnotic suggestion of drinking water. By the same method PLATONOV (1936) induced a tenfold increase in diuresis, but found no change in the hydration of blood. HEILIG and HOFF (1925) demonstrated the effect on diuresis of a hypnotic suggestion of good and bad mood. GROSSMAN (1928) was unable to confirm these data. FÖLDI *et al.* (1956) demonstrated during suggested work in hypnosis changes in haemodynamics and renal function comparable to those caused by actual work.

The principal aim of our experiments was to elucidate the mechanism of the increase in diuresis induced by hypnotic suggestion. Control experiments were first carried out, without water intake and with the ingestion of 500 ml of water. The effect of hypnotic dream on diuresis, was then studied, and an increase in diuresis by suggesting drinking of water under hypnosis was then produced. Finally, after the water diuresis induced by the ingestion of 500 ml of water had ended, an increase in diuresis was induced without delay by suggestion. It was thus possible to compare the mechanism of the two kinds of enhanced diuresis within the same experiment.

METHODS

After careful clinical examination, including tests for renal function, 15 normal human subjects, aged 18–24 years, were subjected to a total of 120 experiments. The test subjects did not eat or drink for from 4–14 hr before experiment, lay down quietly in the same period of the day every day, fully dressed, in a quiet room (17–20°C) and voided at 20 min intervals on instruction by the experimenter. Care was taken that the bladder should be emptied. The urine voided was tested for volume, endogenous creatinine and sodium contents, as well as for osmolar concentration. As their values were but slightly divergent from published data, serum creatinine and sodium were tested on only a few occasions. Endogenous creatinine was estimated according to POPPER, MANDEL and MAYER (1937), sodium was determined in a Zeiss flame photometer (FOLDI *et al.* 1954) and the osmotic concentration was assessed on the basis of the reduction in the freezing point (BALINT and HEGEDÜS, 1955).

It is still debatable whether the creatinine value obtained shows the pure creatinine or the total chromogen value. According to the accepted view in the literature, however, the changes in endogenous creatinine excreation and clearance, respectively, truly reflect the changes in filtration.

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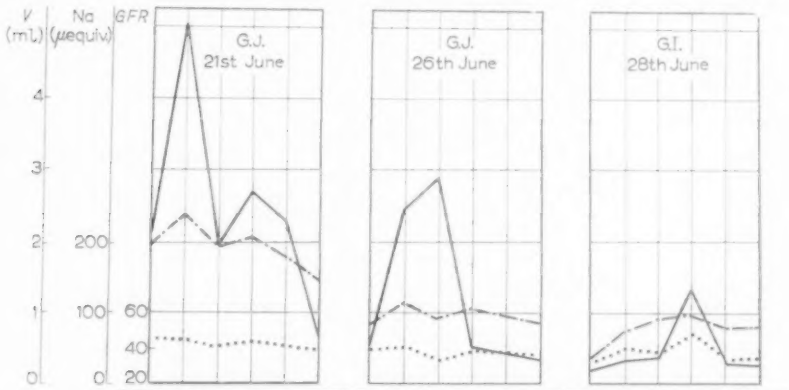
RESULTS

Increase in diuresis in the early phase of the experiment, without water intake

In the early phase of the experiment we have observed the variations in the urine output of the test subjects, who were lying awake and relaxed. Out of the 62 initial experiments on 15 subjects, diuresis significantly increased in 30 experiments on 8 subjects, in spite of the fact that they had been fasting and thirsting for from 4 to 14 hr. The concentration of the urine decreased, then increased, the free water clearance became positive and a diuresis curve similar to that of water



	Spontaneous diuresis G.J. 21st June						Spontaneous diuresis G.J. 26th June						Spontaneous diuresis G.J. 28th June					
	1	2	3	4	5	6	1	2	3	4	5	6	1	2	3	4	5	6
V (V/ml)	2·09	5·02	1·95	2·73	2·53	0·62	0·54	2·47	2·85	0·54	0·47	0·37	0·22	0·33	0·36	1·32	0·28	0·25
U_{Kr} (mg %)	22	9	21	16	16	65	75	17	13	73	86	102	146	120	105	36	113	136
U_{Na} (mequiv/l)	95	47	99	77	71	237	161	47	32	195	208	237	173	151	157	45	146	168
U_{osm} (mosm/l)	275	120	270	205	185	725	715	200	130	675	780	795	940	825	730	265	650	775
G.F.R.	46	45	41	44	41	40	41	42	37	39	40	38	32	40	38	48	32	34
E_{Na} (μequiv)	199	236	193	210	180	147	87	118	91	105	98	88	38	50	57	59	41	42
Na %	2·9	3·5	3·1	3·2	2·9	2·5	1·4	1·8	1·6	1·8	1·6	1·5	0·8	0·8	1·0	0·8	0·9	0·8
C_{osm}	1·88	1·97	1·73	1·83	1·53	1·47	1·26	1·62	1·22	1·19	1·20	0·97	0·68	0·89	0·86	1·15	0·60	0·64
C_{H_2O}	+0·21	+3·05	+0·22	+0·90	+1·00	−0·85	−0·72	+0·85	+1·63	−0·65	−0·73	−0·60	−0·46	−0·56	−0·50	+0·17	−0·32	−0·39

FIG. 1.—Increase in diuresis resulting without water intake in early experiments. (*Above*) the curves. (*Below*) the numerical data (computed for square metre body surface) they were plotted from. Ordinates: V (ml): minute diuresis. Na (μequiv) and E_{Na} (μequiv): sodium excretion. GFR: glomerular filtration rate. U_{Kr} (mg %): creatinine concentration. U_{Na} (mequiv/l.): sodium concentration. U_{osm} (mosm/l.): osmolar concentration. Na %: excreted Na in percent of filtrated Na. C_{osm}: osmotic clearance. C_{H_2O}: free water clearance. Abscissa: 20-min periods; ————: diuresis; .....: filtration; ·—·—·—·→·: sodium excretion.

diuresis resulted. In the majority of cases filtration did not change markedly, and sodium excretion increased during high diuresis.

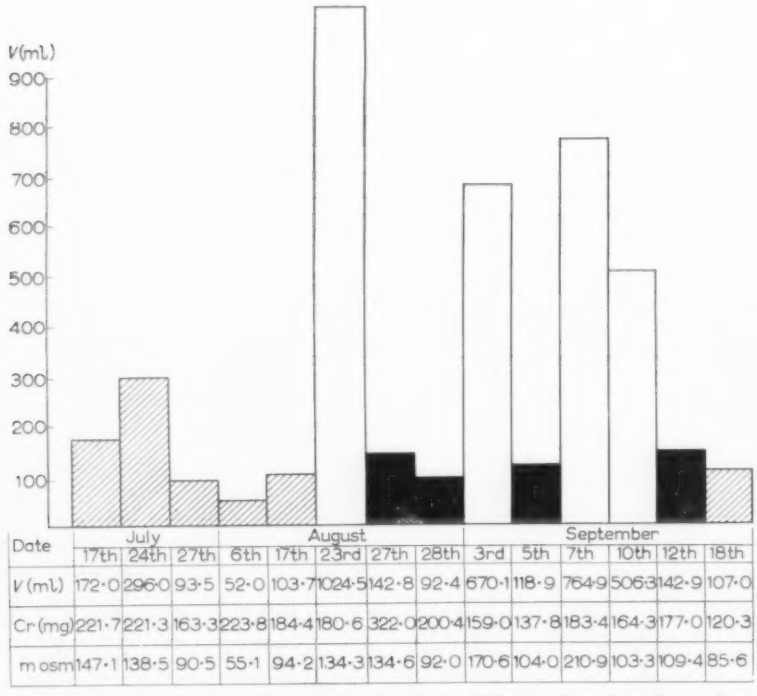
As the experiment progressed the increase in diuresis became smaller and smaller, its peak shifted backwards, and a low, constant diuresis resulted in 3 to 4 experiments (Fig. 1, Fig. 2, 17 July–17 Aug). Subsequently, a spontaneous increase in diuresis was a rare occurrence, except in 2 cases, in which it reappeared after experimentation was resumed after an interval of 4–6 months.

Effect of the hypnotic state

After the spontaneous increase in diuresis had been levelled out, the effect of hypnotic sleep on diuresis was examined. Hypnosis was induced by fixation and by verbal suggestion. Its depth

TABLE 1.—EFFECT OF HYPNOSIS ON THE URINE OUTPUT WITHOUT WATER INTAKE

	Number of experiments	Wake		Hypnosis		Significance (t test)
		Number of periods	V(ml) (20 min)	Number of periods	V(ml) (20 min)	
B. J.	5	10	24·6 ±8·1	13	18·9 ±10·8	$0{\cdot}50 < P$
K. P.	15	28	17·4 ±7·0	26	15·0 ±6·7	$0{\cdot}20 < P < 0{\cdot}50$
K. S.	13	15	23·0 ±2·0	19	21·3 ±5·8	$0{\cdot}20 < P < 0{\cdot}50$
L. L.	9	13	14·9 ±3·9	19	16·8 ±3·5	$0{\cdot}50 < P$
L. M.	4	21	17·0 ±2·1	9	16·9 ±1·1	$0{\cdot}50 < P$



Date	17th	24th	27th	6th	17th	23rd	27th	28th	3rd	5th	7th	10th	12th	18th
V (ml)	172·0	296·0	93·5	52·0	103·7	1024·5	142·8	92·4	670·1	118·9	764·9	506·3	142·9	107·0
Cr (mg)	221·7	221·3	163·3	223·8	184·4	180·6	322·0	200·4	159·0	137·8	183·4	164·3	177·0	120·3
m osm	147·1	138·5	90·5	55·1	94·2	134·3	134·6	92·0	170·6	104·0	210·9	103·3	109·4	85·6

FIG. 2.—Inhibition of water diuresis in deep hypnosis. Columns show the volume of urine excreted in 120 min. Shaded column: waking state, no water intake. Empty column: waking state, after the intake of 500 ml of water. Solid column: deep hypnosis, after the intake of 500 ml of water. Date: the day of experiment. V(ml): urine output, ml/120 min. Cr (mg): creatinine excretion, mg/120 min. mosm: Osmolar excretion, mosm/120 min.

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was controlled by the arm test and by the efficacy of picture suggestion. In 46 experiments without water intake made on 5 subjects no significant change was observed in diuresis after the induction of hypnosis following the initial "wake" periods (Table 1). In 18 experiments the test subject was given 500 ml of water to drink during hypnosis. In the experiments in which the act of drinking was emphasized in the suggestions, a regular increase in water diuresis similar to that seen in the waking state resulted. On the other hand, in the experiments in which the test subject was simply told to drink up the water from the glass held in his hand and drinking was not accompanied by a suggestion of drinking, urine output increased either slightly or not at all, and no water diuresis resulted. In these experiments no substantial changes were seen in creatinine and osmolar excretion, the concentration of the urine did not decrease and the free water clearance (WESSON and ANSLOW, 1952) remained negative throughout (Fig. 2).

Increase in diuresis induced by hypnotic suggestion

In the third series of experiments, 4 subjects underwent control periods in the waking state, and in the hypnotic state and then, while hypnotized, they were given an empty glass to hold, and given suggestions that there was water in the glass, which they drank. In fact, the test subject raised the glass to his mouth and swallowed as if drinking. This procedure was then repeated twice, emphasizing the act of drinking. On questioning after the experiment, the test subject told us that he had drunk 3 glasses of water. There were 16 such experiments.

In 8 cases diuresis increased after suggestion well above the mathematical scattering of the control experiments without water intake ($2s = 2\sqrt{\Sigma(x - \bar{x})^2/(n-1)}$), then returned to the initial value after describing a curve of regular shape. More than once the maximum minute diuresis increased to 10–12 times the control level, the total volume of diuresis (during the 2 hr following suggestion) to three or four fold, or higher than it was in the water diuresis produced by the intake of 500 ml of water.

The suggestion-induced increase in diuresis was accompanied by an increase in filtration, as measured by the creatinine excretion. The concentration of the urine decreases, but the dilution is relatively slight, in spite of the significant increase in minute volume. The urine does not become hyposmotic and, in contrast with what happened in water diuresis, the free water clearance turns even more negative during the increase in diuresis.

The great increase in natriuresis, amounting sometimes to as much as 4–5 times the values observed in the experiments without water intake and in water diuresis, is remarkable. The increase is substantial in relation to the amount of sodium filtrated and also the osmolar excretion significantly increases (Fig. 3, Table 2).

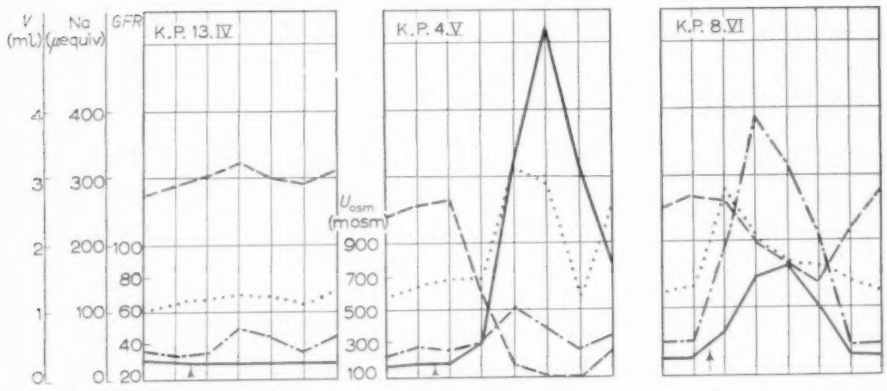
Water diuresis and enhanced diuresis due to suggestion in the same experiment

The mechanism of water diuresis proved to be different from that of the increase in diuresis induced by suggestion. For this reason they were elicited in the same experiment, side by side, on two occasions. The test subject drank 500 ml of water, then immediately after the water diuresis was over he was hypnotised and the drinking of 3 glasses of water was suggested whilst under hypnosis. Thus, the majority of accessory circumstances were identical and the difference between the mechanism became even more obvious.

In accordance with published evidence and the results of the control experiments, in water diuresis resulting in the first half of the experiment the urine is highly diluted, the free water clearance turns positive, filtration slightly increased and natriuresis is almost unchanged. In the second half of the experiment the described pattern of the increase in diuresis induced by suggestion develops. The concentration of the urine is relatively little reduced, the free water clearance turns even more negative, the increase in filtration is moderate, that in sodium and osmolar excretion highly significant (Fig. 4).

DISCUSSION

In the increase in diuresis which occurs in the early phase of the experiments without water intake, filtration shows but insignificant changes. On the other hand, the concentration of the urine is markedly reduced, suggesting that the production



	K.P. 13 April							K.P. 4 May								K.P. 8 June							
	Effect of hypnosis on water output without water intake ↓ Hypnosis							Drinking of 500 ml water under hypnosis with suggestion of drinking ↓ Drinking of 500 ml water								Suggestion of drinking water under hypnosis without water administration ↓ Suggestion of drinking							
	1	2	3	4	5	6	7	1	2	3	4	5	6	7	8	1	2	3	4	5	6	7	8
V (ml)	0·30	0·28	0·25	0·28	0·28	0·28	0·28	0·19	0·22	0·22	0·55	3·17	5·23	3·17	1·70	0·28	0·28	0·69	1·48	1·68	1·02	0·33	0·26
U_{Kr} (mg%)	203	234	262	262	245	232	262	362	337	353	143	46	26	22	73	256	266	172	70	50	84	233	271
U_{Na} (mequiv/l)	148	120	162	270	241	143	244	179	223	187	94	34	14	14	39	197	208	278	295	186	188	138	174
U_{osm} (mosm/l)	1200	1275	1315	1400	1310	1280	1345	1060	1125	1170	600	200	120	95	260	1105	1190	1160	900	725	650	980	1215
G.F.R.	61	66	68	71	69	65	73	69	74	78	79	146	136	70	124	72	74	132	104	84	86	77	70
E_{Na} (μequiv/m)	44	34	42	76	67	40	68	34	49	41	52	108	73	44	66	55	58	192	437	313	192	46	45
Na%	0·5	0·3	0·4	0·7	0·6	0·4	0·6	0·3	0·4	0·4	0·4	0·5	0·4	0·4	0·4	0·5	0·5	1·0	2·8	1·8	1·5	0·4	0·4
C_{osm}	1·18	1·17	1·08	1·29	1·20	1·18	1·24	0·66	0·81	0·84	1·08	2·08	2·06	0·99	1·45	1·02	1·09	2·62	4·36	4·00	2·17	1·06	1·04
C_{H_2O}	-0·88	-0·89	-0·83	-1·01	-0·92	-0·90	-0·96	-0·47	-0·59	-0·62	-0·53	+1·09	+3·17	+2·18	+0·25	-0·74	-0·81	-1·93	-2·89	-2·32	-1·15	-0·73	-0·78

FIG. 3.—Mechanism of the increase in diuresis induced by suggestion. (*Above*) the curves. (*Below*) their corresponding numerical data, in square metre body surface.
– – – –: Osmotic concentration of urine.
Otherwise, the abbreviations and signs are as in Fig. 1.

TABLE 2.—MECHANISM OF SUGGESTION POLYURIA
120 min values. *V*: urine (ml). Na: sodium (mequiv.). K: potassium (mequiv.). Cr: creatinine (mg). Osm.: osmolar excretion (mosm).

		Without suggestion						Suggestion polyuria				
		V	Na	K	Cr	Osm		*V*	Na	K	Cr	Osm
K. P.	4 Exp.-s	87·7 ±19·2	11·3 ±2·0	3·4 ±1·8	155·9 ±21·2	96·5 ±23·2	3 Exp.-s	162·9 ±38·0	28·3 ±14·1	7·0 ±3·1	172·3 ±27·8	127·9 ±38·4
K. S.	16 February	136·0	40·1	5·7	187·3	136·4	17 April	195·5	61·7	7·3	176·3	188·0
	1 March	98·7	35·2	7·0	173·1	115·8	27 April	162·5	50·3	5·0	172·9	150·7
L. L.	21 August	95·3	14·9	1·3	77·3	54·1	16 October	334·0	24·3	2·1	183·3	200·8
	18 October	88·2	5·9	1·2	128·1	73·9	19 October	541·5	28·7	2·6	186·2	358·0
L. M.	5 Exp.-s	99·6 ±9·6	15·3 ±4·9	5·6 ±2·3	178·3 ±19·1	114·1 ±35·4	11 January	195·3	28·7	4·2	193·3	186·6
Total	13 Exp.-s	97·5 ±26·2	15·0 ±12·8	4·1 ±2·2	161·2 ±33·1	102·8 ±24·9	8 Exp.-s	239·7 ±130·7	34·0 ±16·0	5·3 ±2·4	178·8 ±16·9	183·4 ±79·7

Significance, by *t* test: *V*: $P<0{\cdot}01$, Na: $P<0{\cdot}01$ Osm: $P0{\cdot}<01$
K: $0{\cdot}20<P<0{\cdot}50$, Kr: $0{\cdot}20<P<0{\cdot}50$

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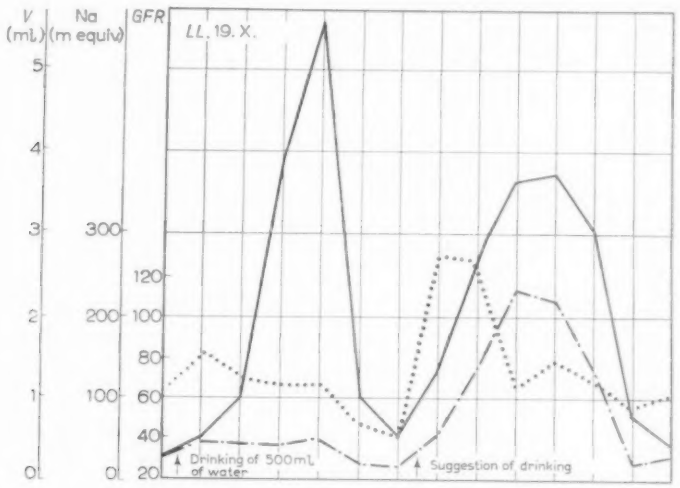
of ADH has decreased. Such a polyuria associated with a dilution of urine has been described as occurring in dogs (GERBNER *et al.*, 1959 a, b); and goats (ANDERSON, 1955) as a result of excitation. A psychical polyuria in excited



	Conscious diuresis after water intake							Hypnotic suggestion of drinking without water intake						
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
V (mL)	0·31	0·52	0·99	3·74	5·58	1·01	0·55	1·35	2·65	3·60	3·73	3·07	0·77	0·46
U_{Kr} (mg%)	209	156	71	18	12	46	75	96	48	18	21	22	70	133
U_{Na} (mequiv/L)	100	93	44	11	9	24	31	40	52	65	60	44	30	60
U_{osm} (mosm/L)	882	834	483	181	123	329	542	599	724	584	646	683	904	1100
G.F.R.	65	81	70	67	67	46	41	130	127	65	78	68	54	61
E_{Na} (μequiv/m)	31	48	44	41	50	24	17	54	138	234	224	135	23	28
Na %	0·3	0·4	0·4	0·4	0·5	0·3	0·3	0·3	0·7	2·4	1·8	1·3	0·3	0·3
C_{osm}	0·90	1·41	1·57	2·22	2·25	1·09	0·98	2·65	6·29	6·89	7·89	6·88	2·28	1·66
C_{H_2O}	-0·59	-0·89	-0·58	+1·52	+3·33	-0·08	-0·43	-1·30	-3·64	-3·29	-4·16	-3·81	-1·51	-1·20

FIG. 4.—Wake diuresis and hypnotic suggestion of drinking water in the same experiment. (*Above*) the curves. (*Below*) their corresponding numerical data, in square metre body surface. Abbreviations and signs as in Fig. 1.

human subjects has been described (EBSTEIN, and SCHWENKENBECHER, 1926; STUTZIN, 1926). Presumably also in our experiments the excitation caused by experimentation in early trials spreads to the hypothalamic centres regulating urine output and this explains the increase in diuresis.

In deep hypnosis, if water intake is not accompanied by a suggestion of drinking, in spite of the ingestion of 500 ml water the urine does not become more dilute and no water diuresis results. Essentially, hypnotic sleep is none other than an inhibition (PAVLOV, 1945). Ample evidence is available showing that a spread of

inhibition, natural and drug-induced sleep inhibit water diuresis. In our opinion in these cases of hypnotic sleep, the inhibition spreads to the hypothalamic centres of water control, diminishing their reaction and therefore, despite the hyposmosis, the production of ADH will be very little reduced or not reduced at all, and the water diuresis will be lower or absent.

In the increase in diuresis induced by suggestion, the increase in filtration is more difficult to evaluate owing to the dead space error and to the lower-than-normal filtration values in low diureses (GERBNER *et al.*, 1959b), but suggests that the glomerular regulation may take part in the propagation of the cerebral cortical effect. Even if it is slight, the decrease in the concentration of urine indicates that tubular water reabsorption is diminished, which, in turn, suggests that antidiuretic hormone (ADH) production is interfered with.

The great increase in natriuresis is due to the fact that the significant increase in urine volume is not associated with a comparable decrease in sodium and osmolar concentration. As in many cases under the influence of suggestion more sodium is excreted at a lower filtration than at the higher filtration of water diuresis, the increase in natriuresis can be ascribed not to an increase in filtration, but primarily to a diminution of tubular sodium reabsorption.

Whereas in water diuresis sodium excretion does not change significantly and the free water clearance turns positive, after suggestion natriuresis greatly increases and the free water clearance becomes more markedly negative. Thus, the mechanism of the increase in diuresis resulting from the suggestion of drinking is different from that developing after water intake, and is similar to what has been described in connection with the diuretic conditioned reflexes in dogs (GERBNER and ALTMAN, 1959). It seems that the cerebral cortical effect is transmitted by similar effector mechanisms.

The nervous regulation of renal tubular function is a much debated topic in the literature (KAPLAN and RAPPAPORT, 1951; BLAKE, 1951; FISCHER *et al.*, 1955; PINTER *et al.*, 1955). The fact that as a result of the effect produced by verbal suggestion words (stimuli acting through the cerebral cortex) tubular sodium re-absorption decreases indicates that what we deal with is a new, as yet unknown mechanism in the nervous regulation of renal function.

SUMMARY

In half of the test subjects an increase in diuresis associated with a dilution of urine developed during the first experiments without water intake. This is believed to be due to a spread of psychic excitation to the hypothalamic centres regulating water metabolism.

Hypnotic sleep does not produce any substantial changes in the diuresis without water intake. In deep hypnosis, after the drinking of water, if the act of drinking is not accompanied by a suggestion of drinking, the urine does not become more dilute and no water diuresis results in many cases. It is believed that in such cases inhibition spreads to the hypothalamic centres of water metabolism, blocks their reaction, and ADH production does not decrease in spite of the hyposmosis.

In the increase in diuresis elicited by the hypnotic suggestion of drinking water the concentration of the urine slightly decreases, suggesting that ADH production has diminished.

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The dilution, however, is relatively small in degree. In spite of the significant increase in volume, sodium and osmolar excretion increase substantially and the free-water clearance becomes more markedly negative. The increased natriuresis is due primarily to a decrease in tubular sodium reabsorption. Thus, as a result of the effect produced by verbal suggestion ADH output and tubular sodium reabsorption decrease.

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AN EVALUATION OF THE ROLE OF EMOTIONAL FACTORS IN MINERS' NYSTAGMUS

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INTRODUCTION

THE causation of miners' nystagmus has been a subject of continued controversy since the condition was first described by DECONDÉ (1861). A variety of aetiological theories have, from time to time, been proposed. The toxic effect of mine gases was suggested as a causative agent by PECHDO (1893), ROBSON (1923) and BUTLER (1939). This theory was refuted by HALDANE (1922) and now has few supporters. Postural factors were implicated by SNELL (1892) who considered that a cramped position together with the maintenance of an upward gaze caused the disability. Similar views were put forward by JEAFFRESON (1887) and REID (1906). A specified labyrinthine origin was suggested by PETERS (1906) and by FAVILLE (1936).

The theory that inadequate illumination is an important causal agent in miners' nystagmus has been held for many years and has been given strong support by the findings of FERGUSON (1939) who, in a controlled investigation, found that the incidence of nystagmus diminished with adequate lighting and was greater when lighting was inadequate. The importance of inadequate illumination in the causation of miners' nystagmus will be referred to later.

That miners' nystagmus may be associated with emotional disturbances or psychiatric symptoms has been observed for many years. The precise role of emotional factors in the aetiology of the condition however, remains very much a subject of controversy. MARTIN (1920) considered the condition to be similar to anxiety neurosis, the main difference being accentuated eye symptoms in miners' nystagmus. CULPIN (1932) studied a series of thirty-six patients and found a high incidence of psychiatric disorders, viz. anxiety states, obsessional states and hysteria. STERN (1948, 1950) also found a high incidence of psychiatric disorders in a series of miners' nystagmus patients. He considered that the disorder was a psychosomatic affection arising from emotional stress but that for the full development of oscillations a prolonged period of work in the dark was also necessary. MURRAY (1951) carried out a detailed analysis of clinical signs and symptoms found in a group of miners' nystagmus patients and concluded that they were mainly neurotic manifestations.

In contradiction to the above findings are the conclusions of BROWN *et al.* (1950) who compared a group of fifty miners' nystagmus patients with a control group of normal coal miners, matched for age and occupation. They found no significant differences in interests, work preference, age at marriage, etc., and concluded that psychological factors were not important. There thus appears to be little agree-

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ment at present on the precise role played by emotional factors in miners' nystagmus. Among many problems, relating to the emotional aspects of miners' nystagmus, awaiting solution are:

(1) The nature, incidence and significance of psychiatric signs and symptoms in miners' nystagmus.

(2) Whether miners' nystagmus patients show a greater predisposition to neurosis than the normal population.

(3) Whether psychogenic influences are a possible primary cause of oscillations in miners' nystagmus.

(4) The relative importance of psychogenic and physiogenic factors in determining degree of disability.

MATERIAL

The medical definition of miners' nystagmus is clear and refers to the development of oscillatory movements of the eyeballs as an occupational disease in coal miners. The regulations governing certification, however, permit a wider definition and enable certification and compensation to be made even when oscillations are not present. In the present study only patients with oscillations were included. The group, attending the Nystagmus Research Clinic of East Glamorgan Hospital, Pontypridd, S. Wales, was a random sample of such patients. A group of fifty patients who had received appendicectomy and herniotomy operations, of similar age distribution, was studied for control purposes.

METHODS

The patients were examined by an opthalmologist and psychiatrist (L. R.) at the clinic. A report on home and social conditions together with an independent history were supplied by the social worker. A special item sheet was completed for each patient. Ratings in a seven point scale were made on various aspects of symptomatology and clinical status. The item sheet facilitated the recording of data and the detailed standardized clinical assessment of each patient. A special electrical method for recording eye movements was used. An improved method of recording eye movements electrically was described by BERGMAN *et al.* (1952). Seven solder disk electrodes are fixed to the skin by cellulose adhesive tape. Six are placed at the margins of the orbits and one placed on the bridge of the nose. These are connected with the input of an electroencephalograph as follows:

Channel 1, right eye. Superior–inferior electrodes, recording vertical movements.

Channel 2, left eye. Superior–inferior electrodes, recording vertical movements.

Channel 3, right eye. Nasal and lateral electrodes, recording lateral movements.

Channel 4, left eye. Nasal and lateral electrodes, recording lateral movements.

According to BERGMAN *et al.* (1952), the deflexions recorded represent changes in the position of the eyes, the amplitude of the deflexions represents velocity and the shape of the individual deflexions the acceleration of eye movements. A large deflexion will, therefore, represent a rapid oscillation and a sharp rise from the base line a rapid change of position.

CLINICAL DATA

The age distribution of the patient group was as follows: 20–30 years 7·5 per cent, 30–40 years 30 per cent, 40–50 years 30 per cent, 50–60 years 18·5 per cent, 60–70 years 11 per cent, 70 years and over 3·6 per cent. The mean age at the time of examination was 46 years.

The age at onset was distributed as follows: 20–30 years 18·5 per cent, 30–40 years 45 per cent, 40–50 years 18·5 per cent, 50–60 years 7·5 per cent, 60–70 years 15 per cent. The mean age at onset was 37 years.

Clinical signs and symptoms found in miners' nystagmus patients

The clinical signs and symptoms commonly found in miners' nystagmus patients can conveniently be divided into (a) ocular and (b) general. The percentage incidence of these features in our group is given in parentheses.

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Ocular signs. In addition to oscillation of eyeballs (100 per cent) there occurred frequent blinking (25 per cent) blepharospasm (10 per cent) and tremors of eyelids (30 per cent).

Ocular symptoms included movements of objects (85 per cent) pain in eyes (60 per cent), photophobia (55 per cent), slowness in dark adaptation (50 per cent), complaint of defective vision (even though visual acuity might be normal on testing) (15 per cent).

General signs. Two special general signs associated with miners' nystagmus are (a) head tremor (b) posture.

(a) Head tremor may be of two kinds: (i) rotary movement of head; (ii) fine tremor which is part of a general increased muscular tension of the body.

(b) The typical posture in miners' nystagmus is the head thrown back to avoid elevating eyes with the hat or cap pulled forward to shade eyes from bright light. Other general signs are somatic manifestation of emotional tension, e.g. tremors, hyperflexia, tachycardia, sweating, dilated pupils, etc., which were found in 70 per cent. Stammering was present in two patients.

General symptoms

(1) The most common symptom was giddiness or unsteadiness, which was most frequently brought on by postural changes such as getting up after stooping or sitting or on turning sharply and occurred in 85 per cent of the group. The patient may stagger or fall and the symptom was found to be particularly distressing.

(2) Headache (52 per cent) was frequently an early symptom and often continued through the illness. The headache is sometimes very severe, sometimes accompanied by nausea and may be either unilateral or bilateral. In our group the frontal and occipital areas were the most frequent sites for headache.

Symptoms due to increased emotional tension or automatic dysfunction occurred in 5 per cent. These symptoms included respiratory complaints, cardiac discomfort, nausea and epigastric discomfort, emotional diarrhoea, palpitations, urinary urgency or frequency, etc.

Other psychiatric symptoms included anxiety, depression, phobias, insomnia, irritability, hysterical symptoms, hypochondriasis, fatigue, etc.

In order to clarify the manifold symptoms and signs found in patients suffering from miners' nystagmus it is desirable to know which are specifically attributable to nystagmus and which are due to other causes. The signs and symptoms specifically associated with, and directly attributable to, the nystagmus or oscillatory movements of eyeballs are the complaint of apparent movements of lights and objects, the typical posture and the type of head tremor described under (a, i) above.

The following ocular signs and symptoms can occur as manifestations of psychiatric disorders unassociated with nystagmus: blepharospasm, lid tremors, slowness in dark adaptation, pain in eyeballs, excessive convergence of eyes, photophobia and impaired visual acuity. Postural dizziness which is often associated with miners' nystagmus is also frequently found in neurotic patients.

The symptoms in Table 1 in the nystagmus group show a striking similarity in distribution to those found in a sample of 15,000 soldiers treated at a neurosis centre (Mill Hill Emergency Hospital) during World War II and in both groups are significantly higher than in the control group. The following symptoms were significantly higher in incidence in miners' nystagmus patients than in the group of neurotic soldiers: postural dizziness, photophobia, spots before eyes, pain in eyes, fear of blindness.

TABLE 1.—INCIDENCE OF VARIOUS SYMPTOMS IN MINERS' NYSTAGMUS COMPARED WITH CONTROL AND NEUROTIC GROUPS

Symptoms	Miners' nystagmus (%)	Neurotic soldiers (%)	Controls (%)
Anxiety and tension:			
mild	24	40	0
moderate	28 } 85	33 } 81	2 } 6
severe	33	8	4
Headaches	52	59	8
Fatigue, lassitude, effort intolerance	52	51	4
Pain, not of demonstrably organic origin, excluding headache	18	25	4
Signs and symptoms of autonomic dysfunction	50	46	4
Irritability	40	33	8

Early symptoms

The most common early symptoms noticed in our group of miners' nystagmus patients were dizziness and/or unsteadiness brought on by (a) postural changes (40 per cent) or (b) other causes (18 per cent) giving a total of 58 per cent, headaches (47 per cent), photophobia (29 per cent) somatic manifestations of anxiety (22 per cent), pains in eyes (18 per cent).

TABLE 2.—INCIDENCE OF PSYCHIATRIC DISORDERS

Psychiatric disorder	Distribution (%)
Anxiety state	52
Anxiety state with hysterical features	7·5
Hysteria	7·5
Depressive state	7·5
Senile dementia	7·5
None	18

It is interesting to note that in a survey of neurosis in soldiers (REES, 1949) the most common diagnostic type of neurosis also was anxiety state which comprised 59 per cent of the total.

Evidence of predisposition to neurosis in miners' nystagmus patients

A relatively high incidence of neurosis and neurotic symptomatology was found in this series of nystagmus patients, and the important question now arises, are persons who develop miners' nystagmus particularly predisposed to develop neurosis?

An attempt to answer this question was made by BROWN *et al.* (1950). They

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compared miners' nystagmus patients with a control group of miners of comparable age distribution and type of work. The survey dealt with items relating to personal interests and hobbies, marital, domestic and social data.

No significant differences were found between the two groups regarding these particular features and the author concluded that miners' nystagmus was primarily physiogenic and that psychological factors were unimportant.

The resistance of some medical authorities to the acceptance of emotional factors in an aetiological role in miners' nystagmus may be influenced by the prevalent idea that neurosis denotes inferiority or the possession of undesirable qualities. As almost everyone is capable of neurotic breakdown if the degree of stress experienced is great enough, the relevant question is not whether a person is predisposed or not, but rather the degree of his predisposition and correspondingly the degree of stress he can withstand before neurotic breakdown. SLATER (1943), from a statistical survey of neurosis in soldiers showed that certain features served as pointers to the neurotic constitution i.e. the predisposition to breakdown with neurosis under stress. The following features were found to be useful indicators of the neurotic constitution:

(1) Family history of psychiatric disorder.
(2) Neurotic symptoms in childhood.
(3) History of previous neurosis.
(4) Clinically abnormal personality.

TABLE 3

	Miners' nystagmus (%)	Normal control group (%)	Neurotic soldiers (%)
(1) Family history of neurosis or mental illness in parents or siblings	15	18·5	60
(2) Neurotic symptoms in childhood	7·5	6	46
(3) History of previous mental illness	0	5·5	10
(4) Personality traits			
Unstable and illadjusted	22	15	56
Timid and meek			
somewhat	11	7·5	38
very	7·5 } 18·5	0 } 7·5	16 } 54
Anxious			
somewhat	26	30	53
very	22 } 48	21 } 51	10 } 63
Meticulous, conscientious, obsessional			
somewhat	22	25	18
very	29 } 51	28 } 53	2 } 20
Shy, reserved			
somewhat	7·5	6	32
very	7·5 } 15	2 } 8	6 } 38
Cyclothymic			
somewhat	15	8	31
very	0 } 15	2 } 10	3 } 34
Markedly hysterical	3·7	0	6
Hypochondriacal			
somewhat	15	4	25
very	3·7 } 18·7	2 } 6	3 } 28

Data relating to these features are given in Table 3 for our nystagmus patients and for the control group, comparable in age distribution and assessed by the same criteria. Data are also given for a group of 1500 neurotic soldiers treated at Mill Hill Emergency Hospital.

Thus two groups (nystagmus and controls) were assessed by one person using identical criteria and the third group of neurotic soldiers by a group of doctors using similar criteria.

The results obtained from comparison of these groups indicate that the miners' nystagmus and normal control groups have a similar incidence and distribution of features indicative of predisposition to neurosis, both groups showing considerably less evidence of predisposition than soldiers treated at a war-time neurosis centre. There is, therefore, no evidence to suggest that miners' nystagmus show a greater predisposition to neurosis than normal controls. This finding is supported by the fact that the group contained many conscientious, hard working individuals who continued to work in spite of constant stress, difficulties and symptoms which had often been present for many years before eventually giving up work. Many men continued at work for many years before reporting the condition, despite their knowledge of its existence.

TABLE 4.—DURATION OF NYSTAGMUS BEFORE REPORTING BY PATIENTS

Duration	Proportion (%)
Less than 1 month	11
1–3 months	3·7
3–6 months	3·7
6–12 months	7·5
1–2 years	26
2–3 years	15·0
3–5 years	18
5–10 years	7·5
10–20 years	7·5

It will be seen that only about one-quarter of the patients reported the condition within 1 year of knowledge of its onset. The factors which motivate against reporting the disorder were usually financial and economic; miners' nystagmus does not tend to develop in the early years of work underground. In our patients the average period of work underground before the onset was 22 years. This finding agrees with that of other workers.

Factors determining time of reporting sick with nystagmus for certification purposes

An analysis of the reasons and motives given for reporting sick with miners' nystagmus after it had already been present for long periods revealed that the most common determinants were emotional in nature or origin. These fell mainly into the following groups:

(1) Fears that personal or other people's safety would be endangered by the condition.

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(2) Fear of blindness.

(3) Anxiety regarding financial and/or employment security and working capacity.

A fear of endangering the individual or others by accidents was frequently engendered by symptoms of dizziness or unsteadiness, whether brought about by postural change or other causes. This was found to be the main determinant for reporting in eleven cases. Three patients reported sick because they feared that the condition would lead to blindness. Six patients reported sick on developing a neurosis which was precipitated by an accident. Six patients reported on developing psychiatric illnesses due to other causes. Some patients reported the condition within a comparatively short time of onset, whereas others, as already noted, knew that they had oscillations of the eyes for many years before reporting sick.

What makes some men report sick soon after the onset of symptoms? There were nine patients who reported sick within 1 year of the onset of nystagmus. In five of these the onset of nystagmus coincided with the development of anxiety states. Two developed a marked fear of blindness, which they thought would result from nystagmus and two were afraid that their symptoms (nystagmus or dizziness) would endanger their safety. Of the remaining two patients one was of low intelligence, and the second of unstable personality with previous behaviour indicative of a marked predisposition to neurosis. The significant fact appears to be that again emotional factors determined the onset of the disability, and the time the condition was reported. In this group such factors came into operation within a comparatively short time after the onset of oscillations.

ROLE OF ACCIDENTS

Six patients of the series (22 per cent) became disabled with miners' nystagmus after being involved in accidents at work. All these patients developed neurotic breakdown following the accidents. The diagnosis was anxiety state in five patients and hysteria in one. Some of these patients had been aware of the oscillations for many years. The accident had precipitated anxiety states and this, together with the fear of further accidents, led to the reporting of the condition. In two patients the accident involved injury to the eyes, one of these having hysterical blindness for three days afterwards.

Temporal relationship between onset of psychiatric illness or emotional disorder and onset of oscillations

In three patients only did the onset of psychiatric illness coincide with the onset of nystagmus. In the remaining patients the oscillations preceded the onset of psychiatric disorder by periods varying from a few months to many years. This indicates that in the majority of patients the oscillations cannot be regarded as being caused by the psychiatric disorder. It must not, however, be concluded that the psychiatric disorder is a result of the nystagmus, as has been suggested by some ophthalmologists. Further consideration will be given to the causal factors determining psychiatric illness when it develops concomitantly with miners' nystagmus and the role it plays in determining the degree of disability.

Can nystagmus be primarily caused by emotional factors? Nystagmus of psychogenic origin has been reported to occur in hysteria and anxiety states and that an enhancement of chronic nystagmus occurs during periods of emotional disturbance (GRANGER, 1953). In our series the large majority had experienced nystagmus for long periods before developing psychiatric disorders or emotional disturbances. Only in three patients was the reported onset of nystagmus contemporaneous with the onset of psychiatric disorder. Thus in the majority of our patients (88 per cent) the available evidence is against the hypothesis that emotional factors are the primary cause of the oscillation in miners' nystagmus. It is possible, however, that emotional

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factors may exacerbate an existing nystagmus. In fact, as GRANGER (1953) points out, it would be surprising if such a highly co-ordinated mechanism involving twelve muscles were not put out of gear by functional disturbances, and in this connexion it is interesting to note that IRONSIDE and BATCHELOR (1945) found evidence of a correlation between muscle imbalance and neurotic constitution.

Eighteen out of twenty-seven of the patients showed clear clinical manifestations of anxiety and tension, and in fourteen these were rated as being moderate or severe in intensity. During the electrical recording of eye movements discussion of emotionally important topics which aroused emotional reactions produced variable results; in some the oscillations were accentuated but in others no significant change occurred.

The role of oscillations in contributing to disability

The findings indicate that the oscillations, *per se*, are not by any means the most important factor determining disability. Field surveys have shown that oscillations can be present in underground coal miners without causing any disability. STASSEN (1919) found nystagmus in 24·2 per cent of 11,000 miners and LLEWELLYN (1912) in 25 per cent. Furthermore the fact that in the present series 61 per cent had oscillations and continued at work for periods ranging from 1–20 years before the development of significant disability indicates that the presence of oscillations is not in itself the main determinant of the disability. The evidence here presented shows that the degree of disability is usually determined to a greater extent by associated symptoms, or by the patients attitude to them, e.g. as when the development of postural dizziness increases fear of accidents.

Role of emotional factors in contributing to disability

The disability in miners' nystagmus is frequently determined by associated symptoms many of which are, wholly or largely, due to psychogenic factors. In three patients, in whom the onset of psychiatric illness coincided with the onset of nystagmus, the main disability was attributable to the patients' psychiatric state and not to the nystagmus. When a psychiatric disorder is present it usually constitutes the major part of the patient's total disability. Emotional factors frequently play an important role in determining the time the patient reports his condition to the doctor. They may also determine certain attitudes which can play an important role in degree of disability, e.g. fear of accidents and/or endangering self and others, fear of blindness or of loss of working capacity.

During the course of the illness, emotional factors may be the main determinant of the duration of the period of disablement and will also influence the success capable of being achieved by rehabilitative measures. The evidence shows that the disability of miners' nystagmus is often mainly attributable to emotional factors, which may not only determine the time of reporting sick, and the onset of significant disability, but the duration of disablement and possibility of rehabilitation and re-employment.

Are psychiatric symptoms the result of oscillations? Some authorities, e.g. BROWN *et al.* (1950, 1954) and CAMPBELL (1948), believe that psychiatric symptoms are a reaction to the oscillations. It is, in fact, difficult to attribute neurotic symptoms to this, firstly because a large number of our patients had oscillations for many years without developing psychiatric symptoms, secondly, this contention would imply a unique aetiology for psychiatric disorders in miners' nystagmus. Psychiatric disorders

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are almost invariably of multiple causation in which individual and environmental causes variously interact. In this series of miners' nystagmus patients, the psychiatric disorders present were, in fact, found to be of multiple causation, with domestic, marital, financial as well as occupational factors playing a part.

In the older patients aged 60 and over, special difficulties contributed, e.g. the gradual falling off of mental and physical capacity and the increasing difficulty in coping with work were particularly important. Definite evidence of senile dementia occurred in two patients. As already observed, accidents, including injuries to eyes, were the main precipitants of neurosis in six patients.

DISCUSSION

It is now established that the development of oscillatory movements of the eyes in diminished illumination can be a physiological phenomenon. A possible underlying mechanism is due to the fact that in diminished illumination the image is visible only in the periphery of the retina. The image is then, by a reflex mechanism, brought back to the centre of the retina where it is invisible. The eye then rotates to bring the image back to the periphery and is again involuntarily brought back to the centre.

Experimental proof that oscillations can be produced by diminished illumination is provided by BROWN (1951) who found that kittens develop severe oscillations if kept in the dark for periods of from 4 to 7 weeks. The cat, like the human, has cones for central vision and rods in the periphery for dark vision. Rabbits and rats do not have this arrangement and do not develop nystagmus if kept in the dark. These findings confirm those of OHM (1916). FERGUSON (1939, 1943), and many other workers support the view that miners' nystagmus is primarily determined by an intensity of illumination below the threshold of perception by cones. Nystagmus itself does not necessarily cause any disability. This is attested by the fact that many of our patients carried on working despite oscillations for many years, and by the results of field surveys which show that many miners have nystagmus without being aware of it. The miner may, in fact, have oscillations with minimal symptoms for many years. He may only have symptoms directly attributable to the oscillations, such as movement of objects and lights. More usually he develops additional symptoms such as headaches, dizziness and various eye complaints, which are the commonest and often the earliest symptoms to develop. These symptoms may still not constitute a significant disability, the miner being able to continue work without great difficulty. If, however, they become severe, or are accompanied by symptoms of psychiatric disorder, it becomes increasingly difficult for the miner to continue, although he may succeed in doing so for a time despite his symptoms, being motivated to keep working for reasons of financial and employment security.

Some symptoms are particularly distressing, e.g. dizziness or unsteadiness arising either spontaneously, or on postural change. This symptom in particular gave rise to fear of accidents involving personal safety or the safety of others. The occurrence of accidents not necessarily attributable to the patient's symptoms, often brings about a change in attitude to symptoms, an emotional disturbance or a frank neurosis, any of which may make the miner report sick. Fear of accidents or blindness, together with the disability due to the psychiatric disorder itself, often determined not only the time of reporting sick, but also the degree of disability incurred.

The findings provide an explanation for why miners are often able to continue

work despite symptoms over long periods, until such time as determined by emotional and psychiatric factors. Our findings indicate that in those who reported a comparatively short time after the development of oscillations became known to the miner, the significant factor was the development of emotional factors a comparatively short time after the onset. In elderly miners the decrease of mental and physical capacity associated with old age increased the feeling of insecurity and made it more difficult to continue work.

The fact that psychiatric symptoms are much more in evidence at the time of certification has also been observed amongst others by BROWN *et al.* (1954). Many authors have incorrectly attributed such symptoms directly to the existence of nystagmus, as mentioned. Sometimes the condition is loosely described as a psychosomatic disorder. This is not justifiable if the term means that emotional factors are causally relevant in producing ocular oscillations.

Similarly many authors refer to miners' nystagmus as hysteria. Hysteria is a disorder in which the patient exhibits symptoms or signs resembling those of organic disease without such being present, for some real or fancied gain without the patient being fully aware of their nature and motivation. Hysterical symptoms usually affect organs or functions wholly or partly under voluntary control. Patients are aware of hysterical symptoms and they are usually the subject of complaint.

Against the view that miners' nystagmus is a form of hysteria are the following facts:

(1) Ocular oscillations may be present without the miners' knowledge.

(2) Oscillations may be present with his knowledge for many years without causing disability or being the subject of complaint and without fulfilling any motive of gain.

(3) The oscillations found in miners' nystagmus are not under direct voluntary control. Patients may be able to bring on oscillation by postural changes, sudden head movements, etc., but these are reflex and involuntary.

(4) Psychiatric investigation of our group afforded no evidence in support of the condition being basically hysteria.

Some patients at a variable time after the onset may develop hysterical symptoms affecting other functions, organs or systems, or may develop an hysterical attitude to their condition or may secondarily utilize symptoms whether directly attributable to ocular oscillations or not for gain. This secondary gain motivation and an hysterical attitude to symptoms can occur in any physical or psychiatric disorder and must not be confused with the disorder hysteria as defined above.

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Finally MURRAY (1951) has never found the type of oscillation seen in miners' nystagmus in any psychiatric disorder. The most common form of psychiatric disorder found in this group was anxiety state. Whereas there was a high incidence of psychiatric illness in the group of certified miners' nystagmus patients, this was not considered to be due to a special predisposition to neurosis in such patients or to marked personality instability or personality type. The group show no greater evidence of predisposition to neurotic breakdown than the control group and considerably less than a group of soldiers treated at a neurosis centre.

The psychiatric illnesses were attributable to a multiplicity of causes, occupational factors being only one of many, whereas there are strong grounds for supposing that poor illumination over a long period is probably the primary factor in miners' nystagmus it is possible that other factors such as labyrinthine stimulation, and postural neck changes which are known to influence nystagmus (BRAIN, 1942) may also be contributory factors.

The degree of disability is often mainly determined by emotional and psychiatric

symptoms which usually determine to a large extent the duration of disability and also the possibility of rehabilitation and the findings enable the following grades of disability in miners' nystagmus to be described:

(1) Oscillations without additional symptoms or disability, the miners continuing at work.

(2) Oscillations with additional symptoms and signs directly attributable to nystagmus, e.g. head posture, but without significant disability and the miner continuing work.

(3) Oscillations with secondary symptoms and neurotic symptoms but able to continue work.

(4) Oscillations with symptoms constituting disability leading to certification.

THERAPEUTIC IMPLICATIONS

The findings of the present investigation have important therapeutic implications. Firstly it is important that the contribution to the total disability by psychiatric and emotional factors should be fully investigated and assessed. Miners' nystagmus patients are best investigated by a team consisting of ophthalmologist, psychiatrist, social worker and general physician. The factors responsible for the psychiatric manifestations need to be ascertained and if possible remedied.

Few patients require prolonged psychotherapy, but all are capable of benefitting from explanation, encouragement and re-education. Group therapy adapted to the special needs of the type of patient has considerable potential therapeutic possibilities. The degree and duration of disability and the tendency to chronic invalidism can be alleviated by simple psychotherapy aimed at an early return to work. The job given on re-employment can even be underground, providing the illumination is adequate and the need for stooping and sudden postural changes is minimal. The work does not have to be light employment as physical exertion is not contra-indicated in otherwise physically fit patients. In long-established or severe cases a period of rehabilitation at a rehabilitation centre might save some men who otherwise might remain unemployable.

The early assessment of the determinants of the disability in the individual patient, followed by prompt treatment, by psychiatric, social or medical measures as indicated, with the aim of early return to work with suitable job placement, should reduce the degree and duration of the disability associated with this socially important occupational disorder.

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THE APPLICATION OF MODERN LEARNING THEORY TO THE TREATMENT OF CHRONIC HYSTERICAL APHONIA

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I. INTRODUCTION

LEARNING theory has already been applied by the present authors to the problem of stammering. A specific case was reported (WALTON and BLACK, 1958) in which the genesis of a stammer was explained by means of a theoretical model. The outcome of treatment deduced from this model suggested that the method was valid and further work on the same lines was encouraged.

The present paper therefore reports a further application of learning theory, this time to a case of chronic hysterical aphonia. A theoretical model is proposed to explain both genesis and chronicity; a method of treatment is deduced; and the results are reported, together with a follow-up of almost a year.

The literature on this type of case is both sparse and conflicting, and would suggest, in the author's view, that the time is now ripe for the validation of a method of treatment based on an experimentally tested model. Thus was the present formulation prompted. In particular, two assumptions were questioned, first that most published work treated acute and chronic aphonia alike, and secondly that the removal of a chronic aphonia, as with any other symptom, has often hitherto been expected to result in the development of an alternative sympton (WALTON, 1958).

The nearest approach to an experimentally verifiable hypothesis concerning differences between acute and chronic aphonia, with any implication for treatment, was proposed by KRETSCHMER (1934): "an hysterical habit . . . is one which began as a voluntary process and has become gradually automatic from repetition . . . hysterical habits include disorders of . . . speech (aphonia)".

Despite KRETSCHMER's earlier recognition of the therapeutic implications behind such differences, HENDERSON and GILLESPIE (1947) appear not to have followed up these implications. Their own suggestions for treatment are partly empirical and partly notional, based on various *ad hoc* theoretical frameworks. The absence of any experimentally founded postulates in their approach precludes any prediction of the likely effects of such treatment. Often, also, it is difficult to decide whether they regard all hysterical aphonics as being homogeneous or whether they recognize differences to exist.

Amongst the methods they advocate are the use of free association, hypnosis, and the use of special methods in the case of patients resistive to psychotherapy. Some confusion arises here because presumably the latter category is more likely to include the chronic case who has not responded to treatment and whose continued aphonia resembles KRETSCHMER's 'hysterical habit'. If this is the case the habit presumably exists in its own right divorced from the original traumatic events which incurred it. It is therefore difficult for the authors to understand HENDERSON's and GILLESPIE's statements, ". . . a patient with a rigid (hysterical) paralysis must be made to move the limb . . . immediate complete success is necessary. . . piecemeal improvement is tedious and often temporary. . . complete function must be restored at one sitting. . . in a proportion of cases the symptom is an hysterical habit which has outlived its usefulness, and which the patient would gladly be rid of if he knew how. *These patients are easily amenable to treatment*". (Yet these are the patients previously described as resistive to psychotherapy.) The words in italics are the writers'.

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MAYER-GROSS and his colleagues (1955), in a later textbook, similarly do not suggest any method of treatment based on experimentally founded postulates. They make certain empirical suggestions for treatment which are open to the same criticisms as those applied to HENDERSON and GILLESPIE. Several methods are raised for consideration. They suggest, for example, that hypnosis or suggestion under narcosis, together with an understanding of the environmental factors in the causation of the illness, might be of remedial value.

The fact that the present patient failed to improve following several years of intensive treatments similar to those suggested by HENDERSON and GILLESPIE and MAYER-GROSS tends both to support the previous remarks against the use of empirical methods in the treatment of such chronic cases, and to indicate the predictive difficulties inherent in using any such *ad hoc* theoretical frameworks.

The present study arose following the suggestion that the patient might respond to a course of treatment based on learning theory.

2. SUMMARY OF CASE-HISTORY

The patient's family background was thought to be relevant to the development of her aphonia, though nothing of significance is reported to have occurred before the age of eleven.

The patient's father left home when she was eleven and was divorced 5 years later. He is reported to have left his wife because she was always stealing. Until the patient (henceforth to be referred to as 'M') reached the age of eleven her father was said to have been distant with her and she felt that she was not wanted by him. On occasions her mother is reported to have made it equally obvious that M was in the way. After M's eleventh birthday, the mother went out to work though she continued to steal. She is reported to have tried to force M into stealing.

A significant incident is said to have occurred when M was visiting her father who was ill. M alleges that on this occasion incest was attempted by her father. The mother meantime continued to steal. About this time M, who was fourteen, was ill in bed with septic tonsilitis. Two detectives visited her and in her room found a quantity of postal orders stolen by her mother from a local Pools firm.

At the age of fifteen M left school and went to work in a wholesale chemists. About this time her mother was working in a Chinese café. Her mother is said to have tried to arrange a marriage between her daughter and the Chinese proprietor for a sum of money. M's grandfather prevented this. On another occasion when M and her mother went to help with the Christmas decorations in the café, her mother conveniently left M in the basement with the proprietor.

Her mother remarried when M was seventeen. M went to live with her mother and step-father. About this time she is said to have had a minor throat operation. When she regained consciousness she found she had lost her voice. A short time after this she was admitted to a neurosis unit for the first time. From early 1951 to 1957 her voice was at best nothing more than a whisper.

Up to the time that M was referred to the Psychology Department for the symptomatic treatment of the aphonia she had been a voluntary patient in the neurosis unit on four separate occasions. By the time of her fourth admittance she had been aphonic for some 7 years and completely mute for 2 of these years. No significant change had occurred in her condition in spite of intensive psychotherapy, hypnosis and several physical methods of treatment which included modified insulin, ether and methedrine abreactions, narco-analysis and LSD. Before treatment began M was examined by the ENT surgeon who expressed the opinion that she would not speak again, although he could find nothing physically wrong with her vocal chords.

3. SYMPTOMATIC TREATMENT OF THE APHONIA

(a) *Proposed theoretical model*

The assumption underlying most published work on hysterical aphonia that the removal of the symptom must necessarily result in the development of some alternative symptom was questioned. It was proposed instead that there are certain cases of chronic hysterical aphonia in which the symptom originally developed as a conditioned avoidance response; that because it satisfied a temporary need it become reinforced; that thereafter it existed in its own right as a habit, and that this habit was divorced

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from the original circumstances which first incurred the aphonia. If such a model is correct then many of the symptoms of which she complained may not have resulted, as did the aphonia, from the original traumatic incident(s) but have resulted secondarily from being aphonic (i.e. from having a symptom or disability which she could not understand or control). The removal of the aphonia would logically result in the dissipation of the other symptoms. Alternative symptoms would then be unlikely to occur.

(b) *Derivation of method of treatment*

A method of treatment was evolved based on: (i) The learning theory constructs of reactive inhibition ($_{I}R$) and conditioned inhibition ($_{I}SR$). (ii) EYSENCK's postulates that those people predisposed to extroverted patterns of behaviour (and thus to hysteric–psychopathic disorders in cases of neurotic breakdown), develop reactive inhibition quickly; and that this inhibition dissipates slowly.

(c) *Method of treatment (phase 1)*

M was required to read from a book into the microphone of a tape recorder. The book chosen was judged by the two psychologists concerned to be basically uninteresting to M. The explanation given was that: "her voice needed systematic exercise and that this could be achieved by her reading, or trying to read, aloud from the chosen book for periods of 15 min at a time. The volume of her voice would then be measured from the tape recording. If she failed either to maintain the volume of her speech throughout the 15 min or to improve the volume on successive 15 min sessions then it would be evident that she was in need of more extensive exercise. An extra 2 min would be added on to each session, in these circumstances, until improvement was made, when the session would be judged to have attained an optimum length for the most beneficial effects of the exercise to be felt. If, on the other hand, improvement were made within a 15 min session, or from one session to the next, then the exercise must be having effect. In order to avoid excessive fatigue sessions would then be reduced by 2 min so long as improvement was maintained."

The above method was adopted on the assumption that, EYSENCK being correct, hysterics should develop reactive and conditioned inhibition very quickly. They would thus become rapidly motivated towards achieving a shorter session.

The reaction potential of the aphonia (i.e. the ability of the patient to be aphonic) would then be determined by the following equation:

$$_{E}SR = (_{H}SR \times D) - (_{I}R + _{I}SR)$$

... where $_{H}SR$ is the habit strength, D the drive, and $(_{I}R + _{I}SR)$ the sum of the reactive and conditioned inhibition. Thus any attempt to use her voice would rapidly result in a considerable amount of fatigue ($_{I}R$). At the same time those stimuli which increasingly became associated with the growth of this fatigue would produce conditioned inhibition ($_{I}SR$). These are the factors which summate to produce the total inhibitory potential ($\dot{I}R$). As this inhibitory potential is increased by M's continual attempts to read, so should the effects of the excitatory potential become counterbalanced (providing the drive level remains constant) until behavioural extinction of the non-adaptive habit (aphonia) occurs. In terms of the equation as $(_{I}R + _{I}SR)$ approaches $(_{H}SR \times D)$ in value, so $_{E}SR$, the resulting performance, approaches zero.

According to this formulation the aphonia can be regarded as a simple learned habit which, in the case under consideration, had reached maximum habit strength. To eliminate the aphonia would

therefore require the building up of a negative habit. HULL (1943) has shown, however, that repetition alone does not lead to the establishment of a habit, either positive or negative, but merely provides the opportunity for learning if a suitable reinforcement is present. In the present case both rest and the reduction of the length of the session were reinforcing agents.

The sessions were carried out at the rate of one per day every Monday, Wednesday and Friday. One psychologist (P.1) only was present for this phase.

Both the writers assessed the change of volume throughout a session by separately listening to the tape recording when played back, and making counts at intervals of the number of sentences, phrases or words which each could succeed in hearing well enough to understand. The tape recorder's volume control was kept at a fixed setting both for "record" and "play" for each session. This volume was somewhat louder than natural voice. Other measures taken to ensure constancy of recording conditions were the placing of the microphone, M, and the book in certain marked positions for the first session and adhering to these positions throughout the experiment.

(d) *Results* (*phase* 1)

The validity of this method of treatment was tentatively established for, within a limited number of trials, M showed rapid improvement. She gave the first evidence of a "voiced" cough during the fourth session, whilst the first certain evidence of the use of her voice was noted in session six. Towards the end of each of the longer sessions however she was hoarse and complained that she felt tired to the extent of being unable to maintain, outside the experimental situation, a comparable level of performance.

(e) *Method of treatment* (*phase* 2)

There were two sessions each day. M was required to read from the same book and her voice was again recorded. Each session lasted for ½ hr. The second psychologist (P.2) was introduced at this stage. During the course of each ½ hr session M was interrupted after 5, 10, 15, 20, 25 min and given encouragement by the experimenter. These interruptions were designed specifically to minimize the fatigue presumed to be associated with the rapid development of the $\overset{I}{R}$ and $\overset{I}{S}R$ in this type of patient.

Now that she had regained the use of her vocal cords it was decided to change the method of the experiment. The necessity for the systematic and controlled exercise of her voice, both to strengthen it and to remove the hoarseness and fatigue when speaking for any length of time, was stressed to M.

P.1 supervised the trials on Monday, Wednesday and Friday whilst P.2 was responsible for those trials on Tuesday and Thursday. P.2 was included so as to introduce M to the idea of speaking normally in a socially expanding group. P.2 was only introduced, however, after the patient had had an opportunity of meeting him several times in an informal way. In this way anxiety would be less likely to occur in the experimental situation when P.2 was present and M would be enabled to cope satisfactorily with this new experience. This is based on WOLPE's principle of reciprocal inhibition (1952, 1954): "If a response incompatible with anxiety can be made to occur in the presence of anxiety-evoking stimuli it will weaken the bond between these stimuli and the anxiety response".

(f) *Results* (*phase* 2)

By and large the improvement shown in phase 1 was continued, despite the greater length of the sessions. However, although several new 'highs' were achieved in the use of her voice, this improvement fluctuated. In spite of the breaks for encouragement every 5 min she appeared very tired at the end of each session, particularly the afternoon sessions. By now her voice was sufficiently audible for the subject matter of her reading to be understood from beginning to end of each session. The ease with which this could be done varied, but contrasted markedly with the earlier sessions of phase 1

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when it was difficult to distinguish even occasional words at the end of 15 min. But perhaps one of the most important results in this phase was that improvement, when it was made, was made irrespective of whether the well-known P.1 or the recently met P.2 was conducting the session at the time.

(g) *Method of treatment* (*phase* 3)

It was now decided that the principle of reciprocal inhibition should be extended so as, firstly, to involve M in speaking within a larger group and, secondly, to endeavour to maintain speech in a more socially realistic situation. Having been at pains to maintain good *rapport* hitherto, it was essential that the new group member should not constitute a source of anxiety to M. M herself was therefore asked to choose a friend from among the other patients. This she did, choosing a young woman whom she had come to know well. With both psychologists present together, the group was now four strong. Instead of the patient reading from a book however, play reading was now adopted. Thus it was hoped to approximate more closely to the conversation of every day life. A comedy was chosen and M was given the part of the character with most lines to speak in whichever scene was being read at the time. Sessions were again of ½ hr duration and were again recorded.

(h) *Results* (*phase* 3)

Although there was now no question that her voice was anything other than completely audible and could be maintained satisfactorily during the play readings sessions, the final sessions of this phase were relatively poorer than those recorded some 10 days previously. The friend also remarked that although M's level of performance with other patients was better than it had been, it was never as good on play reading as it had been on prose reading. Rather than risk possible discouragement, therefore, following the encouragement of the earlier and more satisfactory sessions, phase 3 was abandoned. The cumulative effect of the reactive inhibition generated over the previous sessions was felt to be a possible explanation of this plateau, or failure at this stage to improve further.

(i) *Method of treatment* (*phase* 4).

This phase of the treatment was based on EYSENCK's postulates (1955; 1957):

(i) "Stimulant drugs decrease cortical inhibition, increase cortical excitation and thereby produce introverted behaviour patterns. . . ."

(ii) "Stimulant drugs produce dysthymic symptoms and behaviour patterns and reduce hysterical symptoms and behaviour patterns. . ."

(iii) ". . .individuals in whom reactive inhibition has developed slowly, in whom weak reactive inhibition is generated and in whom reactive inhibition is dissipated quickly are thereby predisposed to develop introverted patterns of behaviour and to develop dysthymic disorders in cases of neurotic breakdown".

On the theory therefore that a stimulant drug should produce an introverted behaviour pattern and reduce the amount of reactive inhibition generated, M was given, to begin with, 10 mg of dexedrine ½ hr before, and later, 15 mg ¾ hr before, each session. Sessions were again of ½ hr duration with the same people present. Play reading continued but often only for part of the session, the remainder of the time being spent on reading by M from the original book to give an up to the minute means of comparison with the speech volume of earlier sessions.

(j) *Results* (*phase* 4)

Slight but definite improvement of voice was noticed during the first session of this phase but more marked still was M's changed attitude. Whereas she had often been lukewarm in her attitude towards the treatment, and sometimes even resentful of the rather punishing situation, under the influence of the dexedrine she became

persistent and enthusiastic. She herself said she felt compelled to do better and felt disappointed when she did not do as well as she would have liked. Improvement continued over the next two sessions and was maintained, also, outside the experimental situation, but she still expressed dissatisfaction with her rate of progress. At the fourth session of this phase the dexedrine dosage was increased to 15 mg, given ¾ hr before the session began. M became even more persistent in her efforts to effect greater improvement whilst her friend, the other patient, reported that during this time there was very little loss of voice during the rest of the day.

At the end of this phase M was given an explanation of the development of her aphonic symptons. She was of high average intelligence and thought to be capable of appreciating this. It was explained to her how it might be possible for a longstanding intractable habit to develop which might easily, through worry and ignorance of its cause, give rise to other additional symptoms. It was pointed out that the symptom would probably have developed originally as a means of satisfying a need and in doing so it would gradually have become reinforced, particularly if the stress situation was long lasting.'

Subsequently to this discussion M was depressed and tearful. The exact cause of this was unknown though it was presumed to have been due to her realization that she might shortly have to return home and find a job. It was suggested to her that it might help her to live away from home. The Psychology Department would carry out a vocational investigation in order to suggest a suitable job. Following this she showed a remarkable change of attitude and a greatly improved and relaxed manner of speaking. She became noticeably very happy and excitedly delighted with her newly found voice. She talked to patients and staff alike without fatigue and without any limits to the amount she spoke.

Attempts were subsequently made, following a vocational assessment, to place her in work in a chemist's shop. Such work was not readily available at the time, so at her own wish she was discharged and left to find work for herself. She obtained employment as a salesgirl in a large Liverpool store in the city centre. She also found herself accommodation.

4. FOLLOW-UP

M's progress was followed up at 1, 3, 7, 8, 10 and 11 months after discharge.

For the 6 months immediately following discharge she worked successfully at the multiple store mentioned above. The store is an extremely busy one and her work at a men's haberdashery counter taxed her voice considerably. One of the writers on visiting the store during the sales, was able to hear M shouting along the counter in order to make herself heard. This was significant as she had been unable to shout at the time of her discharge from hospital. However, apart from tiredness of voice at the end of the day for the first month or so, M showed no other ill-effects and maintained her ability to speak throughout this 6 months period.

After six months M managed to obtain the job in the chemist's shop which she had originally wanted and worked there happily for 3 months.

Since discharge M has had to meet two severely stressful situations neither of which has precipitated a recurrence of the aphonia. Nor has any other symptom arisen in its place. The first stressful situation involved her accommodation. Apparently her landlady's husband would frequently return home drunk at night. Often he would bring his friends and expect his wife and M to entertain them. This did not appeal to M, but she could not avoid the situation by going to bed as the husband would come and wake her and bring his friends to her bedroom. The climax was reached when the husband returned home drunk and threatened both his wife and M with a meat chopper. M went straight to

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her parents' home, where she stayed till she obtained, with the help of the hospital, fresh accommodation. The second stressful situation occurred when walking home one night about a week before the 7 month follow-up. A man attempted to assault her. She ran to the nearest police station, but the man gave up the chase when a bus passed by. Although very distressed she again dealt with the situation without losing her voice.

When M visited the hospital for the 7 month follow-up she was asked to complete the Minnesota Multiphasic Personality Inventory (MMPI). It was considered necessary to have this assessment of her level of adjustment following treatment and the removal of the aphonia, for apart, perhaps, from WOLPE's work (1952) little in the way of objective evaluation has been reported in such circumstances. It was also necessary to verify the commonly held view that the removal of a symptom necessarily results in the development of an alternative. Table 1 shows the results of the four MMPI records completed by M over a period of 4 years. Significantly high scores are in italics.

TABLE 1.—CHANGES IN MMPI SCORES BEFORE, DURING AND AFTER TREATMENTS

Date	*HS*	*D*	*Hy*	*Pd*	*Mf*	*Pa*	*Pt*	*Sc*	*Hyp*	?	*L*	*F*	*K*
14.8.53	65	*89*	*73*	*74*	45	*73*	*81*	*98*	60	50	57	69	40
16.4.57	65	68	75	87	41	59	*79*	*79*	*73*	49	64	66	45
25.4.57	46	57	50	63	41	50	59	60	*73*	48	56	55	44
6.11.57	55	62	66	64	43	64	61	68	60	49	56	61	42

In addition M had been asked to complete the Maudsley Personality Inventory (MPI) immediately prior to discharge in May 1957. This was consistent with the MMPI at the time showing a tendency towards extraversion but neuroticism within normal limits.

At the eighth month a further follow-up was afforded by her admission to a general hospital suffering from bronchitis. Her voice was normal during this period. On discharge her previous accommodation was no longer available so she returned to live with her mother. She gave up her job at the chemist's partly to look after her sister and sister's baby and partly because due to poor physical condition her bronchitis had responded only slowly to treatment. The physicians in charge of her case all regarded the bronchitis as genuine.

The consultant whose O.P. clinic she had occasionally been attending for follow-up reports that her voice was still normal after 11 months. The 11 months follow-up has shown:

(1) There has been no recurrence of the hysterical aphonia.

(2) No alternative symptom has developed in spite of the occurrence of two traumatic situations.

(3) Following the removal of the aphonia test results have both returned to and remained within normal limits.

5. DISCUSSION

In this case a theoretical model is proposed to explain the development of a chronic aphonia; a method of treatment is derived from such a model; an extensive follow-up has been carried out over a considerable period of time, during which the patient has had to meet situations which previously would have aggravated her symptom; and progress has been objectively verified by means of psychological tests. The authors venture to think that this approach marks a development in the application of current psychological procedure to the investigation, treatment and subsequent assessment of the individual psychiatric case.

The significance of the present results is twofold. Firstly its possible relevance to the treatment of other non-adaptive psychiatric symptoms. Secondly its apparent contradiction of some aspects of psychoanalytic theory (following removal of the aphonia there has been no evidence of any alternative symptoms, despite the patient encountering two stressful situations. This might have followed according to psychoanalytic theory).

Support is found for the idea that the aphonia developed originally as a conditioned avoidance response which, because it satisfied a need, became reinforced. The fact that the habit arose at all and the illness became chronic would suggest that the stress was originally long lasting. Because it was long lasting the aphonia would both have become a strong habit and would eventually have existed in its own right independently of the stress situation. Over a period of years, and when the habit had outlasted the original trauma, the habit itself would become the cause for concern. It would have appeared to the patient to be a physical defect, and because of its persistence she would have shown an increase in "neurotic" symptoms. Such symptoms can be regarded in the chronic case as secondary, in that they are the product of the habit and not the other way about as has been thought hitherto in such cases. It is the habit that is primary, and only in the acute stage might one expect to find neurotic symptoms reactive to the stress.

Thus, if the chronic habit can be removed, secondary "neurotic" symptoms will disappear. This in fact happened in the present instance. The absence of any demonstrable neurotic condition following extinction of the habit would then account for the failure of any alternative habit or symptom to develop in place of the aphonia. Such a result also argues against the development of so-called neurotic symptoms as a direct result of any neurotic predisposition. Rather might it argue for the development of symptoms being governed by the interaction of the degree and duration of stress; individual differences in tolerance of this stress, and basic treatment. The introverted neurotic might, for example, tolerate more stress, because the postulated reactive inhibition associated with this stress would tend to build up slowly. The extraverted neurotic would tolerate less stress because the postulated reactive inhibition would build up more quickly. Thus an explanation could be put forward for the introverted neurotic's symptoms (anxiety, depression). Long lasting sources of stress might be tolerated, but, because a solution to the problem is not achieved, anxiety (e.g.) or depression arises. With hysterics less stress might be tolerated, with the result that need-reducing symptoms would tend to develop rapidly. Anxiety and depression would then be less likely to occur.

The demonstration, here, of the independence of an aphonic habit appears of importance in helping to distinguish between the acute and chronic cases, and suggests the particular relevance of learning theory to the treatment of certain chronic symptoms which might be classified as habits.

SUMMARY

A theory is put forward to account for the development of a chronic hysterical aphonia. This leads to the deduction of a method of treatment, of which the application in a specific case is described. The recovery of the patient, whose condition was of 7 years standing, is tentatively taken as vindication of the proposed theory. The success of this method of treatment, based on learning theory, in the face of

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the repeated failure of other methods, leads to the discussion of the aetiology of neurotic symptoms. Conclusions emerge which tend to conflict with much hitherto accepted psychoanalytic theory, chiefly that of the non-replacement of the symptom by an alternative, even after 11 months and in the face of considerable stressful provacation.

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BOOK REVIEWS

L. CHERTOK: **Les Méthodes Psychosomatiques d'Accouchement sans Douleur,** L'Expansion Scientifique Francaise, Paris, 1957. 268 pp.

THIS is a most valuable survey of the psychosomatic methods which have been used since time immemorial to relieve the pain, fear, and apprehension of childbirth. Stimulated by the work of VELVOVSKI and his co-workers in Russia, the Soviet method of psycho-prophylaxis was introduced in 1952 into clinical practice at the Polyclinic of the Metallurgists in Paris. There was also an awakening of interest in the greatly neglected work of GRANTLY DICK READ, whose method of Natural Childbirth was by this time of venerable antiquity. The result is that Dr. CHERTOK has considered the time ripe for a review of the whole subject in the light of the historical development of psychological methods of relieving pain. A bibliography of 458 items, of which many are by Russian workers, provides the background for this scholarly, critical monograph.

CHERTOK begins with an excellent review of the early history of mesmerism in France and in England. French ideas were at that time enthusiastically taken up in England, and some of MESMER's strongest supporters were Englishmen. The compliment was to be repaid later in the nineteenth century, when BRAID's work, so neglected in England, was staunchly upheld and presented to an international audience by AZAM of Bordeaux. By the middle of the century deliveries were being conducted under hypnosis, and the great period of hypnosis (1880–1895) saw a worldwide use of hypnosis in childbirth. After the first World War a new wave of interest was manifest in Germany and Austria, probably arising from the experiences with hypnosis in military psychiatry, and the changing conception of hypnosis. The Russians, too, were not uninterested, and in 1924 PLATONOV and VELVOVSKI addressed the second Congress of Russian Neuropsychiatrists on the use of hypnosis in surgery, obstetrics, and gynaecology. Since then the Russians have developed their methods, so much so that a Ministerial Instruction (from the Ministry of Health) was issued in 1951 laying down the details of the practice of psycho-prophylaxis of the pains of childbirth. By 1953 it seemed likely that at least 300,000 Russian mothers had had their babies using this method.

In England GRANTLY DICK READ had developed a method of childbirth without fear. Much of the book is devoted to a consideration of the English and Russian methods, their theoretical bases, and results. The Russian literature in particular is not easily available, and Dr. CHERTOK has done an invaluable service in collecting and translating the Soviet work. What is even more estimable is the critical detachment with which he treats this literature—a trait not always prominent in reviews of Russian work. He concludes that the work of VELVOVSKI and READ are very similar in practice, although their theoretical orientations differ considerably.

CHERTOK concludes by discussing the problems which face the physician interested in this topic. Pain is clearly of central importance, then comes the question of so-called Natural Childbirth—is there a Natural Childbirth? Psychiatric factors too—what is the importance of anxiety, physiotherapy, and psychotherapy—how do the techniques work? All these questions will continue to intrigue us for the rest of our days—and just as certainly will not be solved in our lifetime.

Dr. CHERTOK has written an excellent book; it must be read by all those interested in psychosomatic medicine.

DENIS LEIGH

S. H. FOULKES and E. J. ANTHONY: **Group Psychotherapy,** Penguin Books, 1957. 263 pp. 3s. 6d.

GROUP treatment as a psychotherapeutic procedure has come to stay, not only because it narrows the gap between the great demand for psychotherapy and the inadequate supply of this commodity, but mainly because it is an adequate form of treatment in its own right which may be specially

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indicated in a variety of clinical conditions. This book on the psychoanalytic approach to the practice and theory of group treatment is therefore very welcome. It is the first book by British authors on this subject, and contains a comprehensive account of the proceedings in therapeutic group sessions, as well as a wide-ranging presentation of theoretical issues. The fact that it appears in the low-priced series of Penguin books makes it available to a wide audience. It certainly deserves to be read, not only by the educated public, but also by doctors and others who are professionally concerned.

The most successful sections of the book are the descriptions in Chapters V and VI of what actually happens in the group treatment of children, adolescents and adults. The imaginative and lively account of group sessions, contributed by a patient who is also an accomplished writer, are vivid portraits of events as seen from the subjective angle of the reporter. One regrets that only a few selected excerpts of this patient's group memoir have been reproduced.

The book is obviously not intended to be a primer for prospective group therapists. This may be the reason why the authors do not mention the difficulties group therapists may encounter, nor the pitfalls that may lie in the path of the unwary. Perhaps it was wise to gloss over these possible complications in a book that will reach the general public. Yet it seems debatable whether it was justified to raise excessive hopes in potential patients by presenting group treatment as a panacea that cannot fail to produce some beneficial effect because the therapeutic process in groups is "spontaneous." The group therapist, the authors state, "assumes that the group, without any pressure from him, will prefer to take the therapeutic road, even though it is a little rougher."

Opinions of this kind flow from the theoretical preconceptions of the authors, who do not mind contradicting themselves when actual happenings conflict with their theoretical postulates. But the discrepancy between theory and reality does at times cloud the issue. It is stated, for example, that psychotherapeutic groups differ from all other groups in that they are given no particular task to perform. Of course, the patients are expected to talk, but this is not talking for talking's sake; this is a "verbal communication" of a special kind; it is, in fact, a "free-floating discussion," because the therapist who conducts the group is so permissive in his attitude that he leaves the "discussion entirely to the spontaneous mood of the group and its members." However, on the very next page, there is the more correct statement that " the conductor accepts and *even encourages* such statements as might not be made in an ordinary social situation" (reviewer's italics). Behind the mask of permissiveness the conductor, after all, conducts and guides the therapeutic group sessions.

These blemishes of vague conceptualization, however, cannot detract from the great intrinsic merit of the book.

F. KRÄUPL TAYLOR

M. SHEPHERD: **A Study of the Major Psychoses in An English County.** Maudsley Monograph No. 3., Chapman & Hall Ltd., 159 pp., 25s.

THIS, the third in a series of monographs reporting work carried out at the Institute of Psychiatry of the University of London is an elegant piece of work. The lack of adequate statistical information in psychiatry has been a stumbling block to progress in spite of the century old recognition of the need for such data. America and Scandinavia have gone some way in remedying the defect, but England has lagged behind. Figures and statistics tend to be dull fare when compared with the living patient confronting the psychiatrist. But large scale morbidity studies have obvious relevance to the growth and development of the psychiatric services, as well as in providing knowledge of the mental disorders themselves.

Dr. SHEPHERD has made a study of the administrative and clinical records pertaining to patients admitted to an English County Mental Hospital in the two triennia 1931–33 and 1945–47. He chose a small county (Buckinghamshire) with an area of about 750 miles², a population of about 400,000 and one antiquated mental hospital serving the county. Comparing and contrasting the patient populations in the two triennia he shows that the pattern of events had very much altered, with a consequent functional transformation of the hospital. Prognosis had improved, the mortality rate had dropped, more patients were coming into hospital and were getting better more quickly. Many other interesting facts came to light, and the monograph is crammed with intriguing information. Dr. SHEPHERD is at

pains to stress that his is a parochial investigation, and that further nationwide studies are urgently required. But his careful and thoughtful study is an example of how such surveys should be carried out. Psychosomatic medicine is in urgent need of similar researches—although in special fields, such as peptic ulcer, much has already been achieved. This monograph can well serve as a model for future surveys of the psychosomatic disorders.

DENIS LEIGH

R. FLETCHER: **Instinct in Man.** George Allen and Unwin, 1957. 339 pp., 40s.

"FLUNG out at the front door, the old instincts are allowed in at the back after assuming an alias and a slight disguise". The author is quoting Professor BURT, and the quotation might well illustrate the standpoint he voices in the book, namely that, until further experimental evidence is available, we shall have to get along with the old concept of instinct. Dr. FLETCHER has examined the evidence, not so much in order to find out how little of it still stands up to critical examination, as—"strongly inclined towards synthesis" —to discover what sort of a workable hypothesis can be constructed out of the evidence and opinions so far put forward.

He has set himself a difficult task, for the broad ecological field in which man moves requires a complexity of adaptive behaviour in which instinctive patterns might well become lost. However, he seeks to define their place in man by a comparison between views put forward by writers of the Ethological School (TINBERGEN and LORENZ in particular) and FREUD's views upon the instincts in man. The book is possibly overweighted by a too lengthy examination of psychoanalytic views. On the other hand, the comparison between the sign-stimuli of the Ethologists and the use of symbols in the social integration of instincts is a stimulating one; so too is the use of the Ethologists' Critical Period Hypothesis in its application to human psychology.

Dr. FLETCHER's most valuable point in the context of contemporary psychological thinking is his emphasis on the influence of affective factors in behaviour. Too little study has been directed towards these because, by contrast with cognitive factors, they are difficult to measure and classify. This does not make them any the less important, however, and Dr. FLETCHER is at pains to emphasize this. Yet few people with clinical experience would agree with this view that the definition between cognitive and affective factors is a false one: one is repeatedly struck by the way in which cognitive powers are preserved even in the severest "functional" mental illnesses. The definition has, in any case, to be made for the purposes of research. Nevertheless, Dr. FLETCHER's study of instinct in man is, in effect, a study of the interplay between these two aspects of mental life, a reminder of the power of affect, and an attempt to redress the balance between that which is easy to measure and that which, though difficult to measure, is nevertheless profound in its influence on human behaviour.

J. P. DEWSBERY

N. WALKER: **A Short History of Psychotherapy in Theory and Practice.** Routledge and Kegan Paul, London, 1957. 185 pp., 25s.

THE author of this pleasantly instructive book is a psychologist who, as chairman of the Davidson Clinic in Edinburgh, works with medical and lay psychotherapists of several schools. He takes his readers on a conducted tour through the practices and theories of many of the leading persons who have influenced the psychotherapies of today. He has a logical bent of mind, and the attraction of his narrative is enhanced by an attempt to compare psychotherapeutic schools according to differences in technique, topics of interest, and explanatory models employed. Two chapters each are devoted to FREUD and JUNG. Another chapter deals with the views of MELANIE KLEIN and ERICH FROMM, and with various attempts at shortening treatment. The practices and thoughts of ADLER and CARL ROGER are presented, as well as the techniques of suggestion applied by the Nancy school and COUÉ. There are also accounts of PAVLOV's views and of the main features of Soviet psychotherapy inspired by them. Methods of group and child psychotherapy also receive attention.

The book is written in an easy style and is likely to appeal to anybody who is interested in psychotherapy and is not too closely wedded to a particular school of thought. The author favours eclectic

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psychotherapists whom he compares to cross-bred cattle, a hardy and versatile breed. But he hopes that the "Freudians and Jungians will preserve their separate and belligerent stud-farms to provide bloodstock for the practical herd of the future".

An interesting table is provided which gives an idea of the popularity of psychoanalysis in fourteen different countries. There are 2754 recognized analysts in the U.S.A.; Britain comes next with 147; all other countries have 60 or less. It can be calculated that there is about one analyst for 60,000 people in U.S.A.; for 80,000 in Israel; for 350,000 in Britain; for 600,000 in Austria; and for 3,500,000 in Germany.

F. KRÄUPL TAYLOR

Ultrastructure and Cellular Chemistry of Neural Tissue. Edited by HEINRICH WAELSCH; with 37 participants. Cassell, London, 1957. (Copyright by Paul B. Hoeber, 1957.) xvii + 249 pp., 40 figures and 32 tables.

THIS book contains the proceedings of a symposium on research with the electron-microscope and cytochemical techniques. Altogether fifteen papers were read and discussed by the participants who were biochemists, anatomists, pathologists and neuro-psychiatrists, among them many of great prominence.

Summary examples may give an indication of the range of the subjects: we learn of the significance of double membranes, as revealed by the electron-microscope; their most fascinating occurrence is in the myelin of peripheral nerve which consists of spiralized lamellae of such membranes developed from Schwann cells (J. D. ROBERTSON). The electron-microscope also disclosed, within the axoplasm of nerve fibres in the neuro-hypophysis, granular material which was identified as the hormone (S. L. PALAY). O. H. LOWRY has amplified his microchemical studies of single cells analysing tissue weighing as little as 0·00002 μg. Cortical neurones of rabbit, monkey and man, grown in tissue culture, continue to metabolize for months in the absence of glia cells, and may even show mitoses in cultures containing cortisone (R. S. GEIGER). A new method for the determination of cell density in different regions of the brain is demonstrated by J. I. NURNBERGER *et al.;* it avoids the shrinkage following fixation and permits of distinction between neuronal and non-neuronal nuclei. Other noteworthy contributions come from G. B. KOELLE who has rendered his histological method more specific for acetylcholinesterase; G. GOMORI on unspecific esterases; A. POPE *et al.*, who find cytochrome oxydase activity greatest in layers 3 and 4 of the cerebral cortex, generally low in gliomas and, interestingly, lowest in astrocytomas; C. N. LOESER, who has developed television scanning as a quantifying method in microspectroscopy, etc.

All papers are of a high standard. The discussion is often of outstanding interest and puts into perspective the significance of new findings as well as the difficulties of techniques and interpretation. Although psychosomatic problems are not directly touched upon, the reader of this journal will welcome a book which, though highly technical, provides him with a cross-section of striking developments in this field, common to the biochemical and morphological sciences.

The illustrations are instructive. The index explains most abbreviations which might otherwise be difficult for the non-biochemical reader.

ALFRED MEYER

T. S. SZASZ, M.D. **Pain and Pleasure. A Study of Bodily Feelings.** Tavistock Publications, London, 1957. xvi + 301 pp., 30s.

ALTHOUGH opinions may differ about the importance and novelty of the views put forward in this book, there can be no doubt about the importance of its subject matter for psychological theory in general, and in particular for psychosomatic theory.

SZASZ begins by clarifying his philosophical position; here he aligns himself most closely with Bertrand Russell's view of the distinction between public and private data, psychology being the science which deals with private data—occurrences which, by their very nature, can be observed by only one person. We should distinguish not between "organic" and "psychogenic" feelings, but according to the point of view from which they are being observed, public or private; that is, from

their physical or psychological aspects. The situation in analytic treatment forces and transmutes data from private to public (though a strictly limited public). The series sensation-perception-affect corresponds to a passage from the realm of physics to that of psychology. Questions framed in terms of sensation will be answered in physically rewarding terms; those formed in terms of affect will be more rewarding psychologically but will tell us little physically (e.g. whether a pain is associated with brain tumour).

The author proceeds to develop a psycho-analytic theory of pain, which he thinks has hitherto been excluded in the theory of the mental apparatus. The essence of his view is that the body is an object for the ego (analogous to external personal objects). Pain can be regarded as a signal, warning the ego of danger threatening the body, just as signal anxiety warns the ego of the danger of losing a needed object. The ego may set up defences against pain, just as against anxiety. The relation between neurotic anxiety and objective fear is paralleled by that between "psychogenic" and "organic" pain—in both cases the differentiation depends on the judgement of an observer. Freud's "economic" pleasure–unpleasure theory is valid only in a closed system and cannot be applied to an object–relationship theory.

The symbolic and social meanings of pain are next considered. During development pain becomes established as a method of asking for help and acquires a communicative meaning. Finally we reach a stage where it is the other person rather than the body that is the main referent. "In all situations where the direct communication of a need is inhibited . . . pain may be experienced and expressed."

Szasz proceeds to discuss states of increased or decreased interest in the body, e.g. in hypochondria and schizophrenia. The hypochondriac is persecuted by his body (object) as the paranoiac by his personal object. Schizophrenia is discussed at some length in terms of object loss leading to taking of the body as the only safe object; attitudes are then transferred from the former external object to the body; finally the body (or parts of it) may itself be lost, and self-mutilation may materialize this loss. There follows an interesting discussion of phantom body parts and phantom pain; the parallel here is between mourning for a lost object, and phantomization after loss of a body part. Phantom pain is equivalent to persecution by a lost object, which happens when there is denial of the body loss and of the painful affect—both return in the form of phantom pain.

The next section of the book deals with the psychology of pleasure. The treatment here is relatively slight—hitherto pleasure has been regarded as self-explanatory, but Szasz shows that the primary model of physiological need reduction is inadequate to cover the immense enlargement in the meaning of pleasure that comes from its association with human relationships and from the concept of pleasure as a substance that must be added to the body or self (cf. pain as a loss). But whereas pain is a command for action, pleasure calls for no action, no change. Hence it, and this section, is of little clinical interest—and hence also so little has been written on the subject elsewhere. Pleasure can in fact be regarded as a signal of safety.

It will be seen that this book is full of interesting ideas; even when not original, they are expressed with much clarity and are well integrated and developed into a consistent system of thought. Szasz's contribution will be very valuable for its clarification of the sometimes rather vague concepts of psychosomatic medicine. His proposed modifications of psycho-analytic theory are more controversial, and this is not the place to discuss them. It is clear that his theory touches closely on Freud's theory of narcissism and Melanie Klein's theory of internal objects—much that he says about the ego's relation to the body would doubtless be expressed by her as a relation to an internal object, identified with a body part. For instance, hypochondria as persecution by an internal object is a very familiar idea, at least in England. It is therefore to be regretted that Szasz has failed clearly to define the relationship between his theory and these others. Perhaps he will do so in future publications.

W. H. Gillespie

ERRATA

Linford Rees (1959) *J. Psychosom. Res.* **3**, 234–241.

In Table 1, on page 236, the word "schizophrenic" in the first column should read "schizothymic".

In Table 3, on page 238, the headings of the second and third columns should be transposed.

In Table 4, on page 240, the figure 45 in the second group of the last column should read 4·5.

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